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TWENTY-FOUR HOUR STRUCTURE OF VIGILANCE UNDER PROLONGED SLEEP DEPRIVATION: RELATIONSHIP WITH PERFORMANCE

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The present study investigated the 36h structure of sleepiness and its relationship with psychomotor performance after 28h of sleep deprivation. Eight subjects, aged 19 to 25 years, participated after spending two adaptation nights in the sleep lab. Subjects came to the lab at 2300 and remained deprived of sleep until 1100. At 1100 a schedule of either 7 min sleep attempt in bed, 13 min awake outside the bedroom, or 7 min resisting sleep in bed, 13 min awake outside the bedroom, was begun and maintained for 36h until

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23h on the next day. The order of the two experimental conditions, which were separated by two weeks, was counterbalanced. Polyhypnographic recordings were carried out during the 7-min "in bed" periods, and psychomotor testing (one and two-handed reaction time tasks) was conducted in the middle of the 13-min wake periods.

In agreement with our previous studies, there were small and nonsignificant differences between the amounts of sleep in each condition. Subjects slept 51% of the time in the attempting sleep condition and 46% of the time in the resisting sleep condition. The structure of sleepiness also agrees with our previous results. There were two sleepiness peaks: a midafternoon peak, and a major nocturnal peak, and two "forbidden" zones for sleep at approximately 1100h, and between 1900 and 2100h. The results of the sleep data are described in great detail in Appendix 1.

Significant circadian effects were found for the two components of the psychomotor performance: reaction time and movement time. The difficulty level of the task only significantly affected movement time, in both experimental conditions. The experimental condition (attempting vs. resisting sleep) had a significant effect on the speed of reaction time and on the stability of movement time. For all levels of tasks' difficulty, performance was poorer in the the resisting sleep condition. There was no interaction however between the level of difficulty and the circadian variations.

In spite of the great similarity between the circadian variations in sleepiness and the circadian variations in performance, correlating these two variables for 12h blocks revealed random and nonsignificant correlations. This negates a causal relationship between the amount of sleepiness and performance, and suggests that both are modulated by a common underlying circadian oscillator. Kaywards?

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1. Introduction

1.1 Fast and Slow Ultradian Rhythms

Spontaneous fluctuations in vigilance levels have been the subject of intensive experimental effort. Most studies have agreed that there are at least two types of fluctuations in vigilance: circadian rhythms in the order of 24h, and ultradian rhythms in the order of 1.5h and 4-5h. Studies conducted in our laboratory in recent years have investigated ultradian rhythms in vigilance and their interaction with the slower ultradian rhythms.

Lavie and Scherson (1981) postulated that ultradian rhythms, if present, should be reflected in subjects' ability to fall asleep at different times during the day. They instructed subjects to close their eyes and to fall asleep during 5 min periods of darkness at 20 min intervals for 12 hours. Sleep and wakefulness were defined electroencephalographically. Significant 1 1/2h ultradian rhythms in sleep stage 1 were evident. As one might expect, ability to fall asleep was also modulated by the circadian rhythmicity, with increased wake time toward the evening hours, and increased sleepiness around midafternoon. Consequently, the 1.5h ultradian variations in alertness levels were much more prominent during the morning.

The rhythmic occurrence of naps during the day was also observed in patients suffering from pathologic somnolence. In a recent study, Volk et al. (1984) investigated the

temporal structure of the diurnal sleep attacks in narcoleptic patients. These patients suffer from uncontrollable naps during the day, sometimes in combination with an inability to control their muscles.. For some narcoleptics, napping occurred cyclically, with a periodicity of about 1 1/2h.

Lavie and Zomer(1983) investigated the relationship between daytime rhythms in the ability to fall asleep and the sleep REM-NONREM cycles. They awakened subjects from either REM or NONREM periods and instructed them, as in the study by Lavie and Scherson (1981) to attempt to fall asleep during 5-min periods of darkness at 15-min intervals for 8 hours. Similar experiments were also carried out without prior sleep between 1600 and 2400. The results of this study showed that the ultradian variations in the ability to fall asleep during the day show two different ultradian components: A 1.5h ultradian component which was found only in the morning experiments and appeared to be synchronized, albeit weakly, with the REM-NONREM cycle, and a second morning component of about 3 1/2h. There were no significant ultradian components during the afternoon-evening experiments.

Evidence for the existence of a second ultradian component in sleepiness that is slower than 1 1/2h was provided by other studies as well. In the second part of the study by Lavie and Scherson (1981) mentioned previously, the effects of sleep deprivation on the ability of subjects to fall asleep at different times of the day was investigated. Under these conditions, the 1 1/2h ultradian rhythmicity was

modified toward a much slower rhythmicity of about 3 1/2-4h. These appeared synchronized across subjects. It should be mentioned that the subjects in the study by Lavie and Zomer (1983) were also partially sleep-deprived because of the early REM and NONREM awakenings which could account for the slow ultradian component detected in that study. Two studies that investigated sleep-wake cycles under the conditions of prolonged continuous bedrest also provided evidence for the existence of slow ultradian cyclicity in alertness (Nakagawa, 1980; Campbell, 1984). In both studies a sleep-wake cycle of about 3-4h was evident although its statistical reliability had not been determined.

Gertz and Lavie (1983) investigated the possible existence of ultradian rhythms in electroencephalographic alertness in yet a different way. They recorded 5-min electroencephalographic activity from 11 subjects at 10-min intervals for 7 1/2h, under baseline and biofeedback conditions. In the latter conditions subjects attempted to either raise or lower the frequency of their EEG with the aid of biofeedback. Both in the baseline and in the feedback conditions there were ultradian rhythms of 7.2 c/d-14.4 c/d (corresponding to periodicitries of about 100 min/cycle) in the mean frequency of the EEG and in the integrated amplitude of the EEG. The biofeedback task had little effect upon the ultradian rhythms with respect to baseline.

Okawa, Matousek and Peterson (1984) investigated the occurrence of spontaneous fluctuations in vigilance, measured by continuous electrographic recordings in subjects who were

encouraged to continue their normal daytime activities. Their results showed that vigilance fluctuations occurred cyclically with periodicities within the wide range of 60 to 110 min. The authors indicated, however, that the fluctuations were more rapid and pronouced during the first half of the day and there was evidence for even longer periodicities in the data. As reported by others, the ultradian rhythms were unstable when within-subjects comparisons were made between experiments conducted several weeks apart. They concluded that the daytime ultradian rhythms may be explained by the simultaneous occurrence of several frequencies.

Hanseau and Broughton (1984) also investigated the occurrence of ultradian rhytms in electroencephalographic activity of awake individuals during the habitual waking day. They recorded frontal and parietal EEGs every 15 min for a duration of 100 sec from eight aduits for 8 hours. Spectral analysis showed the presence of significant 16 c/d (corresponding to periodicities of 72 to 100 min/cycle) rhythms in the total power of the EEG, of each hemisphere. The ultradian rhythms in electrocortical activity were synchronized between the two hemispheres. For individual EEG bandwidths, only the frontal theta showed a marked, though not statistically significant, ultradian rhythm at the expected 16 c/d frequency. In agreement with other studies, a substantial number of spectral peaks were at the slower frequency of 8 c/d, corresponding to periodicities centered at a 3h/cycle.

1.2 Gates of Sleep

The accumulated findings, coming from studies employing widely different methodological approaches, indicate that there are short-term rhythmic variations in the level of alertness during the habitual waking day. Although admittedly these cycles are unstable, having large intra- and inter-subject variability, at certain portions of the habitual waking day they appear remarkably prominent. Furthermore, these special phases which I suggest to call "gates", have important roles in the 24-h regulation of alertness and may have an affect on performance as well. This necessitates a revision of the prevailing views regarding the structure of the diurnal vigilance levels.

It has generally been agreed in the literature that in subjects living under normal sleep-wake schedules, cycles of arousal, synchronized with the cycles in body temperature, reach a peak during the second half of the day, and a nadir during the second half of the night. A more complex function is emerging from our data. Figure 1 depicts a schematic representation of the 24h variations in the probability of a wake-sleep transition (P (W-> S)). The reason for representing the sleepiness rhythms as a probability function is that it provides a quantitative measure of sleepiness which is conceptually easy to understand and practically convenient to measure. Evidentally, the changes in sleepiness across the 24h are comprised of both circadian

and ultradian components.

The ultradian rhythmicity which is superimposed on slow circadian trends reflects the periodic activation of sleep inducing mechanisms. Sleepiness "gates" occur more frequently during the first half of the day, with periodicity close to the sleep REM-NREM cycles, than during the second half, when they are less frequent and less pronounced. The two most distinctive sleepiness gates are the midafternoon and the nocturnal gates, which are about 8h apart (at about 1600 - 2400h). It should also be noted that ultradian rhythms in sleepiness are superimposed on the nocturnal crest of sleepiness as well.

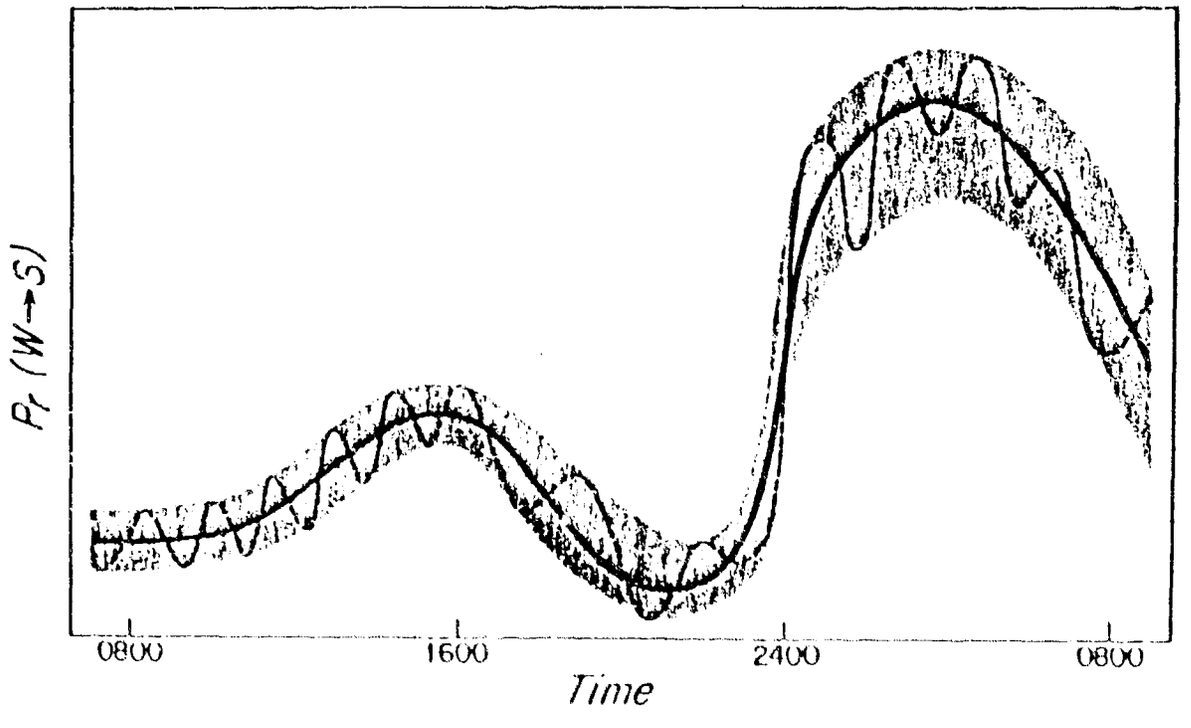

Figure 1: 24-h structure of sleepiness (schematic)

1.3 Effects on performance

The concept of a sleepiness gate is rather new: it implies that there are abrupt physiological changes in the readiness to sleep, rather than an exponential increase in sleepiness assumed by homeostatic sleep theories (Daan et al., 1984) The assumption that the nocturnal sleep gate is switched on as "an all or none" phenomenon raises the important question of how these gates affect human performance. Do the circadian rhythms in performance, which were extensively documented in recent years follow the course of changes in alertness? Is the nocturnal sleep gate coinciding with a comparable "performance gate"? or perhaps the association of alertness with performance is an indirect one, via a third underlying circadian factor. It is possible that while sleepiness is gated as an "all or none" phenomenon, performance is modulated as a continuously changing variable. Another question is the behavioral consequences of the midafternoon secondary gate of sleep. Can we find a comparable dip in performance? The literature about midday variations in performance is far from being decisive. A post-lunch dip in performance was originally reported by Blake (1971) but others reported that the decrease in performance is task dependent, and in some tasks there is a midafternoon improvement of performance (Folkard, 1982).

The purpose of the present study was to investigate the 36h structure of sleepiness and its relationship with psychomotor performance after 28h period of sleep deprivation. The structure of sleepiness was investigated under the conditions of instructing subjects to attempt to fall asleep, and then to attempt to resist sleep.

2. Method and Design

2.1 Subjects - Eight subjects aged 19 to 25 (mean age = 22.6 ±1.76) participated in this study. All were healthy and did not have any sleep related complaints. Subjects had two adaptation nights to the laboratory and then participated in two 36h periods of ultrashort sleep-wake schedule, after 28h of sleep deprivation.

2.2 Experimental paradigm - In each of the experimental periods subjects come to the laboratory at 2300 after having a normal day without naps. They spent the night awake in the laboratory under supervision until 1100. At 1100 a schedule of either 7-min sleep attempt in bed, 13 min awake outside the bedroom, or 7 min resisting sleep attempt in bed, 13 min awake outside the bedroom, was begun and maintained for 36h until 2300 the next day. The order of the two experimental conditions, which were separated by two weeks from each

other, was counterbalanced across the eight subjects. As in our previous experiments (Lavie and Scherson, 1981; Lavie and Zomer, 1983; Lavie, in press) polyhypnographic recordings were performed during the 7-min "sleep and resisting sleep" attempts, and psycho-motor testing was carried out during the 13-min scheduled awake times outside the bedroom (see later). Light snacks and soft drinks were provided every 2 hours throughout the experimental period.

2.3 Performance Measurements - Although we planned to test our subjects on a two-dimensional tracking task, because of various technical difficulties we decided to use the computer-controlled, one-handed and two-handed reaction time task. In this task, after a "go" signal, subjects simultaneously initiate either one-handed or two-handed movements to targets of disparate or equal difficulty and distance. Figure 2 illustrates the experimental apparatus.

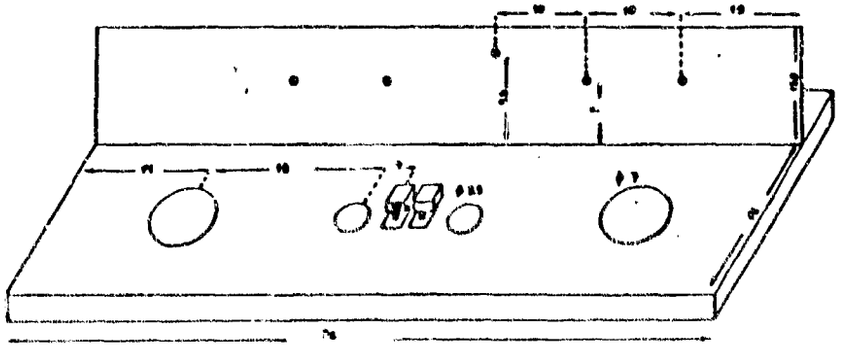

Figure 2: Schematic representation of the experimental apparatus.

It consisted of a plexiglass base (76 cm long, 16 cm wide and 2 cm thick), mounted on a standard table. Two contact switches, centered 0.5 cm apart served as the home keys. The four targets were positioned along the longitudinal center line of the base; two large targets (7 cm in diameter) at a distance of 22 cm from the home keys, and two small targets (3.5 cm in diameter) at a distance of 4 cm from the home keys. A single target was used in the one-handed conditions, and two targets were used in the two-handed conditions. A red light emitting diode served as the warning signal, and similar diodes mounted above the targets served as the "go signal and designated the targets. Stimuli presentation and data collection were controlled by a PDP 11/34 minicomputer.

The subject's task was to move his index finger from the home keys to the target as fast, and as accurately, as possible, after receiving the "go" signal above the appropriate target. The "go" signals were given 1 sec after the warning signal. Five combinations were presented in each 20 min session: 1) right hand "near" target, 2) right hand "far" target, 3) two-handed "near" targets, 4) two-handed "far" targets, and 5) right hand "far" combined with left hand "near" targets. Each of the combinations was presented 5 times with an average intertrial interval of 5 sec. The order of presentations was randomized and two practice trials, which were not included in the final analysis, were presented at the beginning of each session. If the subject missed the target, that trial was excluded and another trial

was presented by the computer.

In each trial, both reaction times (the time from the "go" signal to the release of the home keys) and movement times (the time from the release of the home keys until reaching the targets) were registered by the computer.

From the trials of each combination, mean reaction time and mean movement time were computed. Since there were two one-handed conditions and 3 two-handed conditions, there were 8 separate means for each session. For the purpose of the present presentation, the results were grouped into three levels of difficulty. 1) the mean of all the single-handed responses, 2) the mean of all the symetric two-handed responses, and 3) the mean of the asymetric two-handed response.

The reasons for selecting the coordinated reaction time task are as follows: 1) our previous studies demonstrated that the performance of this task follows the course of the variations in sleepiness, 2) by using both single handed and symmetric and asymmetric two-handed responses, it enables manipulating the level of difficulty of the task and to examine the interaction of task difficulty with sleepiness, 3) dividing the responses into reaction time and movement time, which represent different psychological processes allow the examination of the differential effects of sleepiness on these types of processes.

3. Results

3.1 Amounts of sleep

Six Ss completed the entire experimental protocol. Two Ss, one in the resisting sleep condition and one in the attempting sleep condition, terminated the experiment after 18 hrs (at 0500) because of extreme fatigue. Their data until the breaking point were included. Table 1 presents the mean

Table 1: Mean sleep stages per trial (in mins).

ATTEMPTING SLEEP

Ss	1	2	3-4	REM	total
1	1.07	1.33	0.02	0.01	2.44
2	1.10	1.90	0.00	0.00	3.00
3	1.37	2.06	0.16	0.30	3.89
4	1.39	2.87	0.03	0.02	4.33
5	0.90	2.10	0.21	0.07	3.29
6	0.91	2.69	0.10	0.08	3.80
7	1.01	3.09	0.15	0.12	4.40
8	0.88	1.45	0.11	0.09	2.54
mean	1.08	2.19	0.10	0.09	3.46
sd	0.20	0.64	0.07	0.09	0.76

RESISTING SLEEP

1	0.67	1.36	0.08	0.09	2.21
2	1.13	2.23	0.20	0.00	3.58
3	1.33	1.83	0.02	0.10	3.29
4	1.55	2.37	0.05	0.00	3.97
5	0.98	2.72	0.18	0.10	3.99
6	0.85	1.42	0.00	0.03	2.32
7	1.35	2.63	0.00	0.16	4.15
8	0.96	3.20	0.31	0.36	4.85
mean	1.10	2.22	0.10	0.11	3.54
sd	0.29	0.64	0.11	0.11	0.90

percentages of sleep stages 1, 2, 3-4 and REM for each of the subjects for the two experimental conditions. As in our previous experiments there were only marginal and nonsignificant differences in the amounts of sleep between the "sleep" and "resisting sleep" conditions. Subjects slept 51.4% of the total time allocated for sleep in the "sleep" condition, as compared to 48% of the total time in the "resisting sleep" condition. Four subjects had more sleep in the "sleep" condition, while 4 subjects had more sleep in the "resisting sleep" condition. For more information on the structure of sleepiness see Appendix 1.

3.2 Performance

3.2.1 Effects of Level of Difficulty

Table 2 presents the mean reaction times and mean movement times separately for each of the eight different responses, and for the three levels of difficulty (single-handed responses, symmetric two-handed responses and asymmetric two-handed responses) grouped together. A repeated measures ANOVA (subjects x level of difficulty x blocks) was performed to determine if the level of difficulty had a significant effect on performance. For the purpose of this ANOVA, trials were grouped into 6 blocks, of 6 hours each, or of 18 trials.

Table 2: Mean reaction times (RT) and movement times (MT), in msec, for the three levels of difficulty for the 6 blocks of trials.

Resisting sleep

Block	single-handed		symm. two-handed		asymm. two-handed	
	RT	MT	RT	MT	RT	MT
1	442.7	163.4	436.0	184.3	446.6	219.9
2	467.2	175.5	463.0	212.2	474.3	243.6
3	552.5	233.4	551.4	276.2	563.9	307.2
4	553.1	224.3	540.5	268.9	555.6	309.2
5	520.2	208.9	505.6	245.6	522.9	286.3
6	494.3	209.8	494.9	255.2	507.6	293.0

Attempting sleep

	RT	MT	RT	MT	RT	MT
1	451.2	164.1	443.9	194.0	461.4	236.9
2	452.9	175.6	441.4	208.0	462.2	256.4
3	535.9	225.7	526.4	273.5	534.1	312.7
4	527.7	213.3	522.9	263.8	529.0	304.9
5	488.2	198.3	480.9	251.2	496.8	298.5
6	458.6	199.4	447.9	229.8	463.5	287.9

Reaction time - For both attempting sleep and resisting sleep conditions, the ANOVA revealed significant main effects of subjects and blocks (for both, $p < .0001$), but there was neither a significant effect of difficulty level, nor a significant interaction of difficulty level x Blocks. Fig. 3 and 4 present the mean reaction times for each of the 6 blocks for the two experimental conditions.

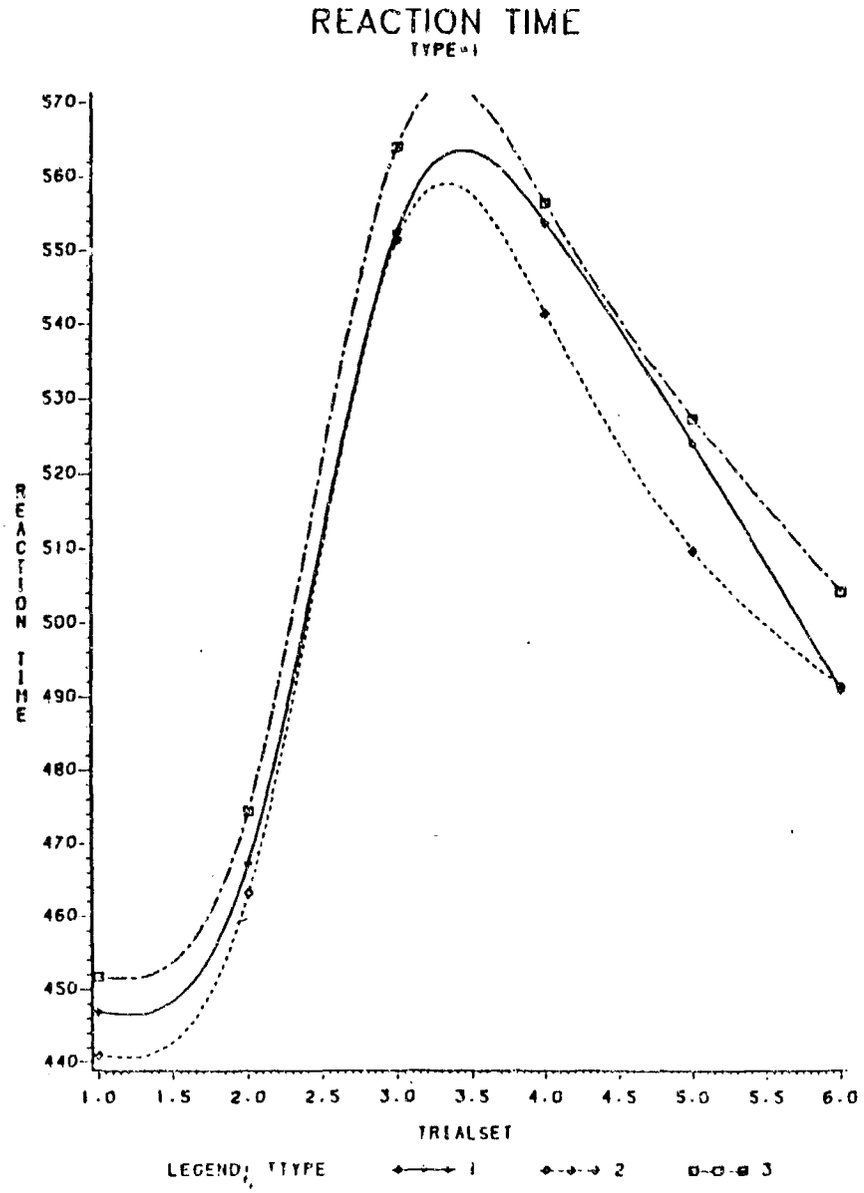

Fig. 3: Mean reaction times for each of the 6 blocks for the three levels of difficulty (1 - single-handed, 2 - symmetric two-handed, 3 - asymmetric two-handed) for the resisting sleep condition.

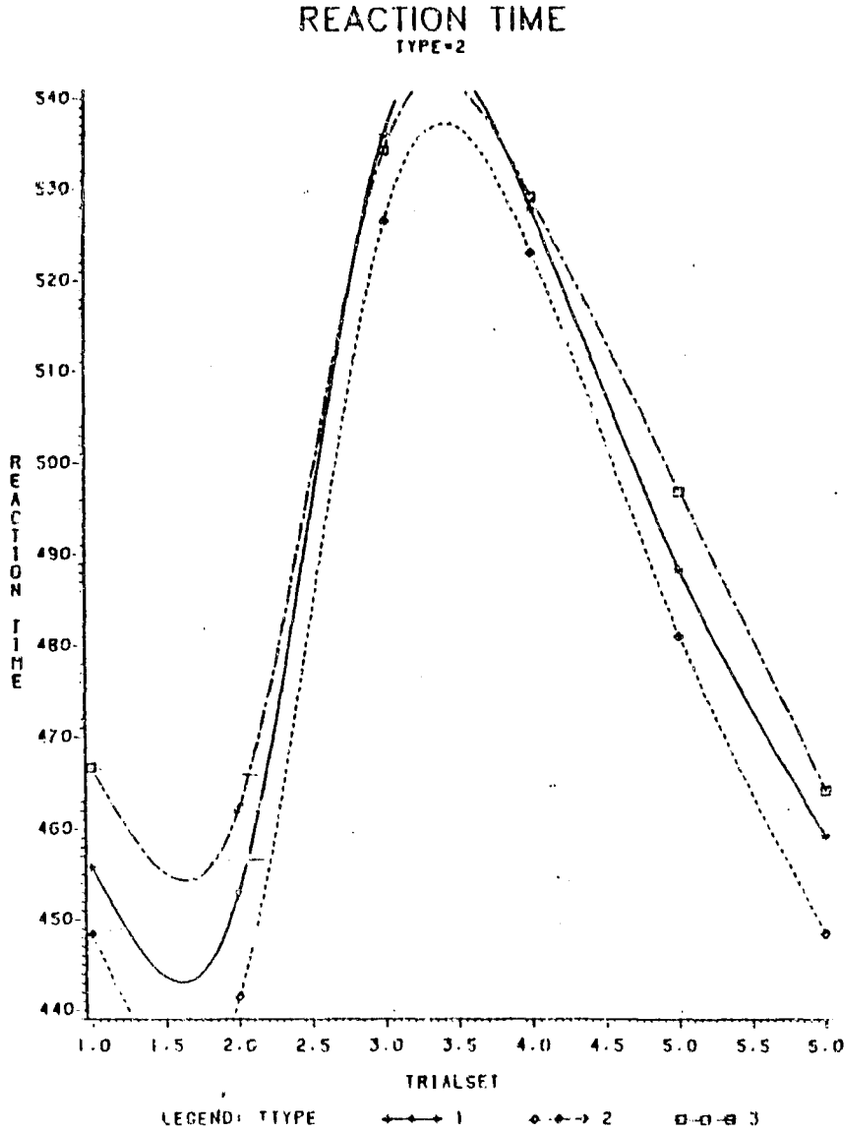

Fig. 4: Mean reaction times for each of the 6 blocks for the three levels of difficulty (1 - single handed, 2 - symmetric two-handed) 3 - asymmetric two handed) for the attempting sleep condition.

The circadian effect is very clear. Post hoc Duncan's multiple range tests organized the 6 blocks in the resisting sleep condition into 4 groups: Group 1 included the first block, Group 2 - the second block, group 3 - the fifth and the sixth blocks and Group 4 - the third and the fourth blocks.

The organization of groups in the attempting sleep condition was slightly different. Group 1 included the first, second and sixth blocks, Group 2 - fifth block, and group 3 - the third and fourth blocks. Thus, unlike the resisting sleep condition in which subjects level of performance during the last 6 hours was significantly lower than during the first 12 hours, the last 6 hours level of performance in the attempting sleep condition was not significantly different from the level of the first 12 hours. This indicates a stronger circadian effect in the attempting sleep condition, or a lesser sleep deprivation effect.

Movement time - Unlike the nonsignificant effect of the level of difficulty on reaction time there was a significant effect of difficulty level on movement time, in both experimental conditions. In addition, there were significant main effects of subjects and blocks (for all $p < .0001$). Post hoc Duncan's tests revealed significant differences between each pair of difficulty levels, for both experimental conditions. Movement times of the two-handed asymmetric responses were significantly longer than the two-handed symmetric responses and the single-handed responses. The two-handed symmetric

responses were significantly longer than the single-handed responses. Figs 5 and 6 display the movement time data.

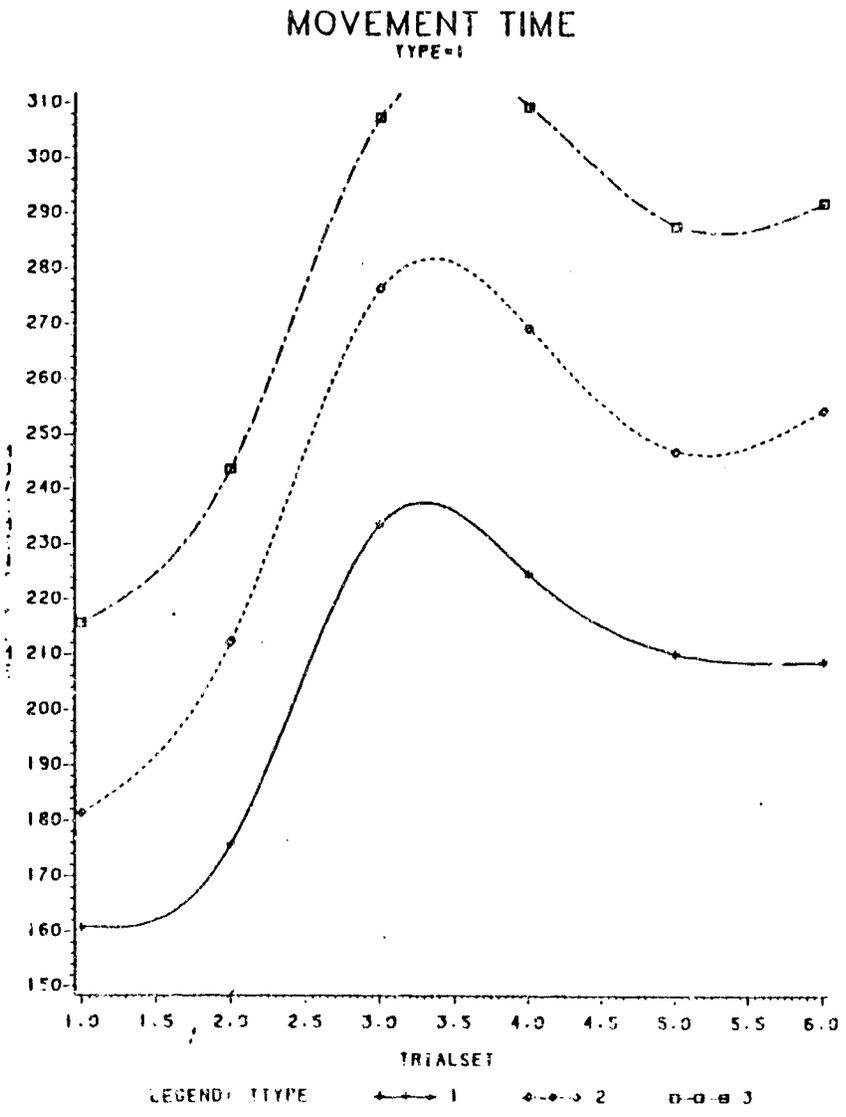

Fig. 5: Mean movement times for each of the 6 blocks of trials for the three levels of difficulty (1 = single-handed, 2 = symmetric two-handed, 3 = asymmetric two-handed) for the resisting sleep condition.

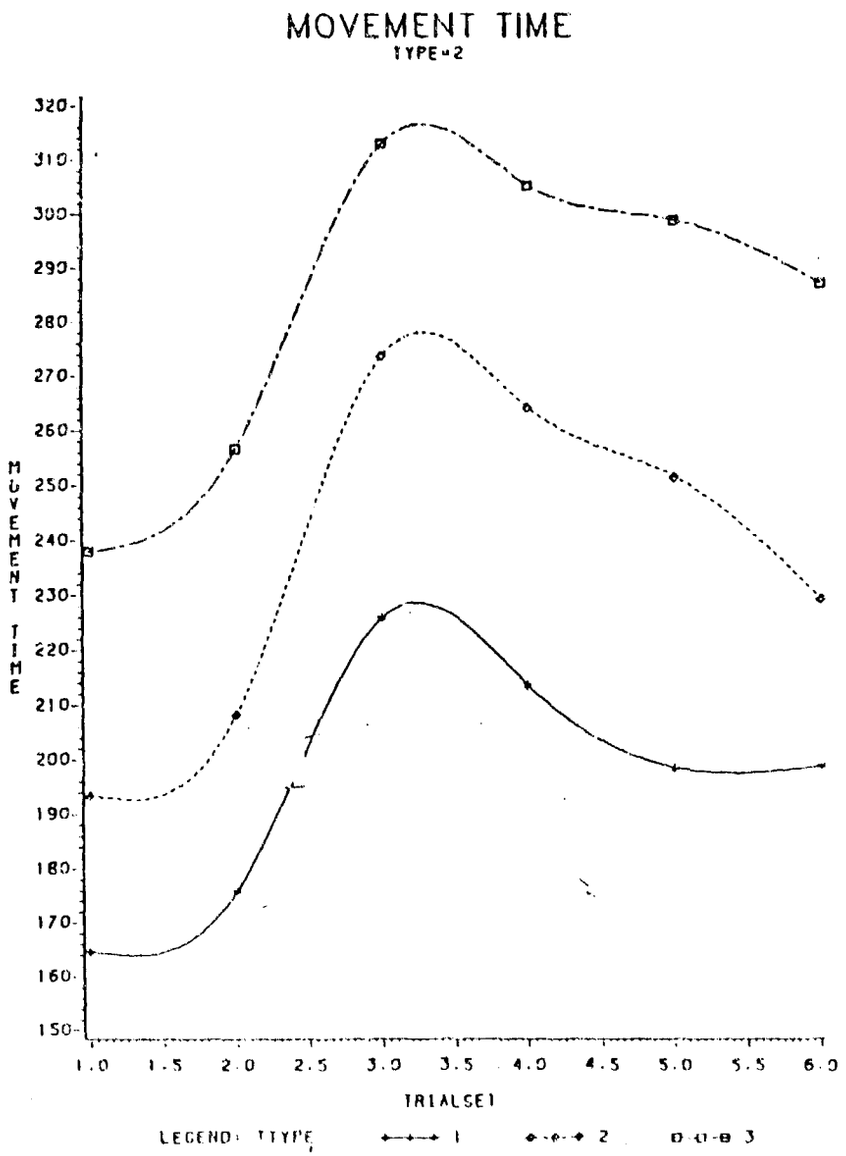

Fig. 6: Mean movement times for each of the 6 blocks of trials for the three levels of difficulty (1 = single-handed, 2 = symmetric two handed, 3 = asymmetric two handed) for the attempting sleep condition.

These Figures clearly demonstrate the lack of interaction between level of difficulty and blocks. The second and third levels of difficulty caused a constant increase in movement time above the single-handed performance level, which remained the same across the entire 36-h experimental period.

The 6 blocks were organized by the Duncan's tests in a similar way to the blocks organization for the reaction time data in the resisting sleep condition. Group 1 included the first and second blocks, group 2 - fifth and sixth and group 3 - the third and fourth blocks.

3.3 Interaction between experimental condition and performance

Since the level of difficulty had a significant effect on movement time, the possible effect of the experimental condition ("resisting" vs. "attempting" sleep condition) was assessed for each level of difficulty, separately.

Reaction time - A repeated measures ANOVA (subjects x block x sleep condition) revealed significant main effects of Subjects, Blocks and Experimental condition, for each one of the three levels of difficulty. In each level, resisting sleep performance was worse than attempting sleep performance. The significance levels were $p < 0.07$ for the single-handed responses; $p < .009$ for the symmetric two-handed responses; and $p < .04$ for the mixed responses (Table 2)

Movement Time - None of the corresponding ANOVAs for movement time yielded significant F ratios. Thus, although there were no significant differences between the amount of total sleep obtained in each of the experimental conditions, instructing subjects to resist sleep resulted in poorer reaction times, but did not affect movement times.

3.4 Performance stability

Visual observation of the data revealed that in spite of the lack of significant differences between movement times in the two experimental conditions, there was a large difference in the stability of performance. This was confirmed by statistical analysis. ANOVAs performed on the standard deviations (calculated over all 18 trials in each block) revealed significant between-conditions differences only for the movement time data. For two of the three difficulty levels, the standard deviations in the resisting sleep condition were significantly larger than in the attempting sleep condition. The significance levels were $p < .03$ for the single-handed responses, and $p < .04$ for the symmetric two-handed responses. Therefore, although there were no significant between-conditions differences for movement time, performance in the resisting sleep condition was significantly less stable than in the attempting sleep condition.

3.5 "Gates" of Sleep and "Gates" of Performance

To determine the possible effects of the sleep gates on performance levels, we correlated the timing of the nocturnal "sleep gate" with the timing of the "switchover" in performance from below to above the 36-h mean. "sleep gate" was defined in the following way: The first resisting or attempting sleep trial after 1900 with at least 50% sleep which was followed by at least 5 out of 6 trials meeting the same criterion. A "switchover" in performance was defined as the first trial in which performance changed from below to above the 36-h mean followed by at least 8 out of 10 trials in the same direction. Since only 6 Ss completed both conditions, to increase the number of subjects, the data of 4 Ss who participated in a similar experiment from 0700 until 0700 the next day, were also included.

Fig.7 presents the scatter diagram for the sleep gates and the performance switchover for the two experimental conditions, for the movement time and for the reaction time data. It can be clearly seen from the figures that there was a strong linear relationship between the two events. Subjects with delayed sleep gates, also had delayed switchovers in performance. The Pearson product moment correlations between the timings of the sleep gate and the switchovers in performance were all highly significant (at least $p < .01$).

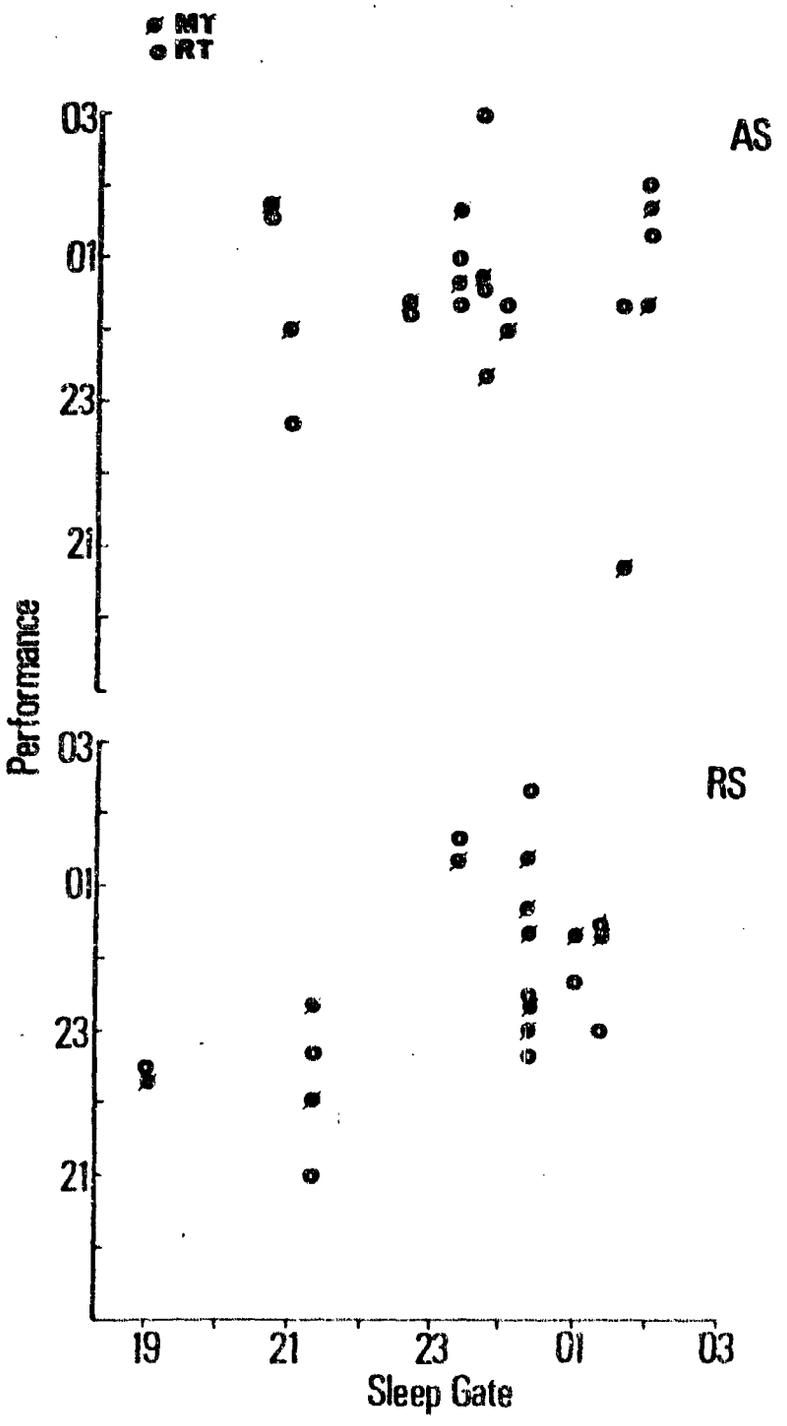

Fig. 7: Scatter diagrams for the sleep gate and the performance switch-over times for the Attempting Sleep (AS) and Resisting Sleep (RS) conditions, for Movement Time (MT) and Reaction Time (RT) data.

4. Discussion

The design of the present study required subjects to repetitively perform a psychomotor task for an extended period of time, after a 24-h period of sleep deprivation, while obtaining only partial amounts of sleep. In spite of the accumulated sleep loss, performance did not progressively deteriorate but instead showed a marked circadian modulation. The appearance of the circadian modulation was evident in both experimental conditions of attempting sleep and resisting sleep, and in both aspects of performance, reaction time and movement time.

Reaction time and movement time reflect two different cognitive processes. Reaction time, the time elapsing from the warning signal until releasing the home keys, reflects selective attention processes. It was assumed to be independent of task difficulty level (Kelso, 1979). Movement- time, the time from releasing the home keys until hitting the targets, reflects the retrieval and execution of specific motor programs necessary to perform the specific movement. Movement time most probably involve short term memory processes, and was shown to be affected by task difficulty.

The assumption that reaction time and movement time are differentially linked to variables related to task difficulty is clearly supported by the present results. Analysis of variance revealed that movement time was significantly affected by task difficulty. The times to execute the two-

handed asymmetric responses were significantly longer than the execution times of the single-handed responses and the symmetric two-handed responses. Also, the execution times of the symmetric two handed responses were longer than that of the single-handed responses. In contrast, task difficulty level had no discernible effect on reaction time.

Based on previous results of the effects of sleep deprivation and circadian rhythmicity on performance, an interaction between task difficulty and circadian effects was expected. Johnson(1982) predicted that a difficult task, which involved memory-related processes, would suffer more from sleep deprivation than simpler tasks. The present data do not support this assumption. There was no significant interaction between task difficulty and hour of testing. This was clearly evident from Fig. 5 and 6. The variations in movement time in each level of difficulty paralleled each other with no evidence for a larger modulation of the most difficult task in comparison with the most simple task. Thus, it appeared that at least with respect to psychomotor tasks which involve a retrieval of preprogrammed motor schemes, the widely held view that simple tasks suffer less from sleep deprivation than difficult tasks should be reconsidered.

The close similarity between the the variations in total sleep and the variations in performance suggested a causal relationship between the two variables. This was also supported by the tendency toward an overall significant positive correlations between total sleep and performance. However, the suggestion that the similarity between the

amounts of total sleep and performance indicates a direct dependence of performance on sleep should be rejected. Removing the circadian effect by calculating the sleep-performance correlations for 12-h periods, yielded few significant correlations, mostly for the 12-h night period (23-1100). This leads to the conclusion that at least during the day period there was no relationship between sleep propensity and performance, which was independent of the circadian effects. It suggests, instead, that both the amount of sleep and the level of performance have their own independent oscillators, which are synchronized by the same circadian zeitgebers. The persistent correlation during the night period might reflect a common linear trend of increasing amounts of sleep and progressively deteriorating performance.

The suggestion that the circadian cycles in performance are independent of the circadian cycle in sleepiness was also made by Wever (1982). He showed that under light-dark conditions the circadian rhythms in sleep-wake and performance could be separated from each other.

The temporal correlation between the sleep gate and the performance gate was rather remarkable. Both performance components, reaction time and movement time switched from below to above the mean in close proximity to the individual sleep gate. This synchronicity may be explained in several ways. First, it could suggest a masking effect of the sleep gate on performance. That is, although there was no overall correlation between the circadian rhythms in sleepiness and

that in performance, there was an immediate effect of opening the sleep gate on performance. The fact, however, that in some cases the switchover in performance actually preceded the sleep gate, makes this explanation unlikely. Second, it is possible that both circadian cycles obeyed the same enviornmental zeitgebers (time givers), such as changes in light-dark cycle, feeding times, etc. This explanation can also be rejected. Although the enviornmental conditions were common to all subjects, each had his own preferred sleep gate which was stable over a two week period. If indeed the changes in sleepiness and performance were induced exogenously, then a more uniform timing was expected. Thus, we remain with the third possibility that both variables were entrained to the same underlying factor, which may be called a circadian rhythm in arousability. It should be emphasized once more that the postulated underlying circadian cycle in arousability should not be equated with thecircadian cycle in sleepiness measured in the present study. The latter can be seen as a cycle of sleep propensity reflecting the probability of making a wake to sleep transition (see Fig. 1 in Introduction).

The lack of a significant correlation between the amount of sleep and performance during the first 12-hr indicates that the secondary mid-afternoon sleep gate does not have any behavioral consequences. It also precludes the possibility that the nadir in sleepiness occurring just before the sleep gate was associated with any improvement in performance. This dissociation between the structure of sleepinesse and

performance during the first 12 hr indicates that although both variables were entrained by the same underlying circadian factor, the control mechanism of sleepiness is much more complex than that of performance. Thus, in conditions of sustained operation under a prolonged sleep deprivation condition, unless subjects actually fall asleep during the midafternoon period, no decrement in perceptual-motor performance should be anticipated.

The emerging differences between the effects of the two experimental conditions of attempting to fall asleep and attempting to resist sleep on performance are interesting and important. Although subjects obtained exactly the same amounts of sleep in the two experimental conditions, the specific instructions had differential effects on performance. Subjects performed significantly worse under the resisting sleep condition. Reaction times were significantly slower and movements time were significantly less stable. These results indicate two things. First, although unsuccessfully so, subjects indeed invested an effort in order to remain awake; second, the invested effort came on the account of performance level. Thus, under the same sleep deprivation conditions operators attitude toward sleep i.e., avoiding sleep as much as possible, or getting some sleep whenever possible, might have a profound effect on the operator proficiency level.

Another point of interest is the differential effects of the experimental conditions on the two behavioral components which comprised the total response. While attempting to

resist sleep actually decreased the level of reaction time performance, it only affected the stability of movement time. This fact and the fact that only movement time was affected by the task difficulty level support the previous assumption that each relys on different cognitive processes which are differentially affected by subjects attempt to resist sleep. As previously mentioned, reaction time reflects processess involving selective attention while movement time involves short term memory and activation of preprogrammed motor schemes. It is possible that when resisting sleep subjects consciously, or unconciously, searched for subjective sleep sensations and sleep related signs which could enable them to anticipate impeding sleep. This constant search for sleep indicators was particularly intensive when subjects were in bed attempting to resist sleep. Most probably, however, this searching activity continued outside the bedroom during the 13-min waking periods. Thus, it can be suggested that the allocation of attention to search for sleep signs interferes with other attention demanding tasks. Since movement time did not depend on attention level it was not affected.

The destabilization of movement time by the attempts to resist sleep can be explained in the following way. Once the home keys were released the speed of the movement was dependent upon two factors: first, retrieving the specific motor scheme from memory storages, and second, its proper activation. Both types of operations are automatic and are relatively independent of attention level. Thus, the instability in movement time can be seen as occasional

failure either to retrieve the specific motor scheme or a failure to activate it. Such a failure could occur, for instance, if a "sleep sign" was detected concomitantly with the motor scheme activation. This suggestion is further supported by the fact that the most significant effect was not found for the most difficult response level of the asymmetric response combination, but for the easier single-handed and the two-handed symmetric responses.

5. Appendix 1: "GATES" AND "FORBIDDEN ZONES" FOR SLEEP

5.1 Introduction

Insight into the laws that govern the timing of sleep and waking has been mostly obtained in environments free of time cues. Isolating humans from all geophysical zeitgebers releases the endogenous sleep-wake cycle from its periodic environmental constraints. In such conditions sleep and waking continue to replace each other periodically with period lengths longer than 24 hrs (for an extensive review see Wever 1979). In most of these studies, naps, or short sleep episodes other than the major nocturnal sleep episodes, were either not allowed, or were eliminated from the data before final analysis. Implicitly at least, the waking phase of the 24 h sleep- wake cycle has been considered to be homogenous with respect to the level of arousability.

This highly idealized assumption was refuted, however, in studies investigating daytime alertness levels using continuous recording techniques, or fast sampling rates. These provided evidence that the level of alertness during the waking portion of the 24 h sleep-wake cycle varies in a cyclic manner with periodicities centered at about 1.5 h (Kripke 1972; Lavie 1979; Lavie and Scherson, 1981; Oertz and Lavie, 1983; Lavie and Zomer,1984; Manseau and Broughton, 1984). Such a daytime cyclicity was previously hypothesized by Kleitman (1963) to be a part of an ongoing 24-h cyclicity

(The Basic Rest-Activity Cycle hypothesis). In addition to the evidence for the existence of 1.5-h cyclic variations in alertness, there is a convincing evidence of a midafternoon dip in alertness which is unrelated to lunch time and represents an endogenous diurnal nadir in alertness (Webb and Agnew 1977; Carskadon and Dement 1979; Lavie and Scherson 1981). Broughton (1975) suggested that this hypoarousal period might be a part of a 12-h cycle.

In recent years we have utilized an ultrashort sleep-wake schedule to trace the course of the daytime variations in sleepiness (Lavie and Scherson 1981; Lavie and Zomer 1983). These studies confirmed the existence of 1.5-h ultradian rhythms in sleepability and indicated that the cycle has more than a single frequency. Furthermore, sleep deprivation appeared to modify the diurnal structure of sleepiness toward slower frequencies.

In the present series of three experiments an ultrashort sleep-wake paradigm was employed to study the structure of sleepiness during the entire 24-h period, and to study the effects of sleep deprivation on this structure. In addition, the effects of contrasting experimental demands: instructing subjects to attempt to fall asleep and instructing subjects to attempt to resist sleep, were investigated. The reason for investigating sleepiness under both experimental conditions was to determine if the temporal structure of the ability to fall asleep is different from the temporal structure of the ability to resist sleep. A similar structure would support the interpretation that the 24 h

variations in sleepiness are regulated by an active modulation of underlying hypnogenic structures. On the other hand, different temporal structures would weaken this interpretation and would suggest instead the possible influence of factors related to subjects acquired sleep wake-habits. The present paper describes the basic structure of sleepability and wakeability under these experimental conditions in three different experiments.

5.2 EXPERIMENTAL PROCEDURE - METHOD & DESIGN

5.2.1 Experiment 1

Six healthy young adults, aged 22 to 26 yrs old, with no complaints about sleep, were paid to participate. Each spent a night in the sleep laboratory from 2300 to 0700 for habituation. During that night electrodes were taped in place, but no recordings were performed. During the experimental periods, subjects came to the laboratory at 1800, after having a normal day without naps. They were fitted with electrodes to record EEG, EOG, and EMG. At 1900, they began a schedule of 7-min sleep, 13-min awake, for 24 hrs. Every 20 min they were instructed to lie in bed in a darkened sound-attenuated bedroom, and attempt to fall asleep (Attempting sleep condition - AS). Electrophysiological recordings were carried out during the 7 min sleep attempts to determine the sleep stages. At the end of each 7-min

trial, whether asleep or awake, subjects were requested to leave the bedroom. At the middle of the 13-min scheduled wake periods, they were tested on one-handed and two-handed coordinated reaction time tasks. Results of the behavioral testing will be reported in a separate publication. Approximately equal size meals of light snacks and soft drinks were available every two hours throughout the 24-h experimental regimen.

The second part of the experiment which investigated the temporal structure of subjects ability to resist sleep (Resisting sleep condition - RS), was conducted about 2 weeks later. As in the first part, subjects came to the laboratory at 1800, were fitted with electrodes, and at 1900 began an ultradian schedule of 7-min awake in bed with eyes closed, 13-min awake outside the bedroom, for 24 hrs. The specific instructions to the subjects were to lie in bed with eyes closed, and try and resist sleep for 7-min Electrophysiological recordings were performed during the 7-min RS trials as before. Here, also, at the end of the 7-min trials, whether asleep or awake, subjects were taken outside the bedroom and were tested on the same tasks.

The order of experiments was counterbalanced across subjects. To motivate subjects to conform to the experimental demands, extra monetary bonuses were paid to the best 2 performing subjects in each condition.

5.2.2 Experiment 2

The second experiment investigated the effects of sleep deprivation on the 24-h structure of the ability to fall asleep and on the ability to resist sleep. Eight healthy male subjects, aged 23 to 26 yrs, were paid to participate. None had any complaints related to sleep and all had experienced sleep deprivation previously. Each spent a habitual night as in Experiment 1. During the experimental periods subjects came to the laboratory at 2300, after having a normal day without naps. They spent the night in the laboratory, awake, under close supervision of an experimenter. At 0700, they began a 7-min sleep, 13-min wake schedule, as in Experiment 1, for 24 hrs until 0700 the next day. Two weeks after the first part, they completed the second part of RS condition. The order of experimental conditions was counter - balanced across subjects.

5.2.3 Experiment 3

The purpose of Experiment 3 was to determine if extending the sleep deprivation period by 4 hrs, until 1100, and extending the ultrashort sleep-wake period by 12 hrs, would affect the temporal structure of alertness. Eight subjects aged 22 to 26 yrs were paid to participate. None had any complaints related to sleep and all had experienced sleep deprivation previously. Each spent a habitual night as in Experiments 1 and 2, and two experimental periods. During the

experimental periods subjects came to the laboratory at 2300 after having a normal day without naps. They spent the night in the laboratory awake under the close supervision of an experimenter. At 1100 they began a 7-min sleep, 13-min awake schedule as in Experiments 1 and 2, for 36 hrs until 2300 on the next day. Two weeks after the first part they completed the second part of the RS condition. The order of experimental conditions was counterbalanced across subjects.

5.2.4 Data analysis

As in our previous studies (Lavie and Scherson 1981; Lavie and Zomer 1984), each of the 7-min trials was scored for sleep stages 1, 2, 3-4 and REM, according to Rechtschaffen and Kales (1968).

5.3 Results

5.3.1 Sleep stages

Tables I-III present the mean amounts of sleep stages 1, 2, 3-4 and REM per trial for each subject in the two parts of the three experiments. There were no significant differences between the RS and AS conditions for any of the stages. Furthermore, in experiments 2 and 3 which involved sleep deprivation, total sleep was slightly higher in the RS than in the AS condition, 4.01 vs 3.7 min in experiment 2, and

3.54 vs 3.46 min in experiment 3. Generally, in all three experiments total sleep was mostly comprised of sleep stage 1 and sleep stage 2. The amount of stage 1 was remarkably stable across the three experiments and the two experimental conditions, overall mean was 1.0 ±.3 min. In contrast, there was a large between-subjects variability in the frequency and amounts of sleep stages 3-4 and REM. Each of these stages accounted for less than a mean of 15 sec per trial.

As could be expected there were significant group differences in the amount of total sleep. In both conditions, more sleep was obtained in experiments 2 and 3 which involved sleep deprivation, than in Experiment 1 which began at 1900 after a normal day. The highest amount of sleep was obtained in Experiment 2 in the RS condition (mean = 4.01 ± .69 min). The same group slept a mean of 3.7 ± .47 min per trial in the AS condition. The least amount of sleep was obtained by subjects of group 1, 3.02 ± .98 min in the RS condition, and 3.26 ± 1.11 min in the AS condition.

Analysis of variance confirmed the existence of significance between-group differences in the amount of total sleep. Since each of the experiments was conducted for a different length of time, and during a different circadian phase, the ANOVA was performed for the time period 1100 to 1900, which was common to all groups. An analysis of variance for a 3 x 2 x 9 mixed design with two repeated measures was computed for total sleep per hour (i.e., the mean of total sleep in three consecutive trials). The factors were experimental group, experimental condition and hours

experimental condition (AS or RS) and hour of day being repeated measures. There were significant main effects of hour ($F=2.83$, $df=7,2$; $p<.007$) and group ($F=8.69$; $df=21,3$; $p<.007$), and a significant interaction of groupxhour ($F=2.22$; $df=14,3$; $p<.007$). The significant interaction is explained by the fact that at 1100 the three experimental groups were sleep deprived to different degrees, and therefore showed different time-related trends in sleepiness.

5.3.2 Temporal structure of sleepiness

In spite of the significant differences in the amount of total sleep, the three groups had a remarkably similar temporal structure of sleepiness. Fig 1-3 present the mean sleep histograms for the AS and for the RS conditions, for the three experiments. In each, there was a major nocturnal sleep episode consisting of 20 to 25 consecutive trials with a sleep latency (to the first 30 sec of sleep stage 2) of no more than 1.5 min, and a secondary midafternoon sleepiness peak at around 1600. These were evident in both experimental conditions. In experiments 2 and 3, there was a nadir in sleepiness at around 2000 to 2200, under both AS and RS conditions. Subjects of group 2, who began the ultrashort sleep-wake schedule at 0700 after 24-h of sleep deprivation, showed initially elevated amounts of sleep.

Table I :Mean sleep stages per trial (in mins) for the subjects in Experiment 1 investigated from 1900 to 1900 without prior sleep deprivation.

ATTEMPTING SLEEP

Ss	1	2	3-4	REM	total
1	0.98	0.77	0.01	0.02	1.79
2	0.57	1.52	0.13	0.00	2.22
3	1.30	1.86	0.00	0.08	3.25
4	0.89	3.13	0.20	0.07	4.31
5	1.33	3.09	0.13	0.04	4.61
6	1.37	1.90	0.08	0.00	3.37
mean	1.07	2.05	0.09	0.03	3.26
sd	0.31	0.92	0.07	0.03	1.11

RESISTING SLEEP

1	0.78	1.15	0.00	0.00	1.93
2	0.66	1.60	0.19	0.00	2.46
3	0.97	2.92	0.03	0.17	4.10
4	0.85	2.78	0.37	0.21	4.25
5	1.01	2.13	0.00	0.00	3.14
6	0.82	1.29	0.09	0.01	2.22
mean	0.85	1.98	0.11	0.06	3.02
sd	0.12	0.75	0.14	0.10	0.98

Table II: Mean sleep stages per trial (in mins) for the subjects in Experiment 2 investigated from 0700 to 0700 after one night of sleep deprivation.

ATTEMPTING SLEEP

Ss	1	2	3-4	REM	total
1	0.72	2.23	0.49	0.00	3.45
2	1.04	3.04	0.38	0.05	4.54
3	1.20	2.52	0.23	0.19	4.18
4	0.75	2.68	0.19	0.05	3.69
5	0.69	3.54	0.07	0.01	4.32
6	2.00	2.70	0.06	0.10	4.58
7	1.11	3.12	0.11	0.06	4.32
8	0.77	1.90	0.00	0.02	2.70
mean	1.04	2.72	0.19	0.06	4.01
sd	0.43	0.51	0.17	0.06	0.69

RESISTING SLEEP

1	0.98	2.43	0.38	0.00	3.80
2	1.21	2.45	0.04	0.04	3.75
3	1.53	2.64	0.20	0.08	4.46
4	0.78	2.53	0.07	0.00	3.39
5	0.82	2.16	0.14	0.02	3.16
6	0.85	3.28	0.00	0.13	4.28
7	0.48	2.68	0.05	0.00	3.22
8	1.68	1.69	0.11	0.07	3.56
mean	1.04	2.48	0.12	0.04	3.70
sd	0.40	0.45	0.12	0.04	0.47

Table III: Mean sleep stages per trial (in mins) for the subjects in Experiment 3 investigated from 1100 to 2300 after 28 hours of sleep deprivation.

ATTEMPTING SLEEP

Ss	1	2	3-4	REM	total
1	1.07	1.33	0.02	0.01	2.44
2	1.10	1.90	0.00	0.00	3.00
3	1.37	2.06	0.16	0.30	3.89
4	1.39	2.87	0.03	0.02	4.33
5	0.90	2.10	0.21	0.07	3.29
6	0.91	2.69	0.10	0.08	3.80
7	1.01	3.09	0.15	0.12	4.40
8	0.88	1.45	0.11	0.09	2.54
mean	1.08	2.19	0.10	0.09	3.46
sd	0.20	0.64	0.07	0.09	0.76

RESISTING SLEEP

1	0.67	1.36	0.08	0.09	2.21
2	1.13	2.23	0.20	0.00	3.58
3	1.33	1.83	0.02	0.10	3.29
4	1.55	2.37	0.05	0.00	3.97
5	0.98	2.72	0.18	0.10	3.99
6	0.85	1.42	0.00	0.03	2.32
7	1.35	2.63	0.00	0.16	4.15
8	0.96	3.20	0.31	0.36	4.85
mean	1.10	2.22	0.10	0.11	3.54
sd	0.29	0.64	0.11	0.11	0.90

REM periods appeared in all experimental conditions. In some subjects its appearance resembled the periodic REM-NONREM cycle in uninterrupted sleep periods. The dynamics of REM sleep in the ultrashort sleep cycle schedule will be described in a separate publication.

5.3.3 'Gates' and 'Forbidden Zones' for sleep

Although the average sleep histograms (Figs. 1, 2 and 3) give the impression of a gradual increase in the amount of nocturnal sleep, examination of the individual histograms revealed that the onset of the nocturnal sleep period was abrupt, almost an 'all or none' phenomenon.

These are exemplified in Figs. 4 and 5 which present individual histograms of a representative subject. As can be seen, the onset of the nocturnal sleep period was abrupt and occurred at approximately the same time in the two experimental conditions.

To investigate the stability of the timing of the onset of the nocturnal sleep episode, a sleep gate was defined in the following way: the first trial after 1900 containing at least 50% sleep of any stage, which was followed by at least 5 out of 6 consecutive trials meeting the same criterion. Except for 2 subjects, one in experiment 1 and one in experiment 3, both in the RS condition, a distinct sleep gate was identified in this way for each of the rest of the 42

experimental periods. Examples of clearly defined nocturnal sleep gates are demonstrated in Figs 4 and 5, which present individual histograms. The timing of the gates was remarkably stable. Fig. 6 displays the relationship between the timing of the onset of the nocturnal sleep period in the AS and in the RS conditions, in the three experiments; these varied from 2100 to 0400 and from 2100 to 0430, respectively. The mean onset times (±Sd) for the AS and RS conditions were 0016 ± 2h 13 min and 0056 ± 2h 30 min, respectively, in experiment 1; 2330 ± 1h 50 min and 2358 ± 2 min in experiment 2, and 2312 ± 1h 36 min and 2306 ± 1h 36 min in experiment 3. The differences between the mean onset times in each of the three experiments were not statistically significant. Twelve subjects showed an earlier gate in the AS than in the RS condition, 7 subjects showed the reverse, and 2 subjects showed no difference. The overall mean difference (27.6 min) between the timing of the sleep gates in the two conditions only bordered on statistical significance ($t=1.44$;, $df= 19$; $p<.1$).

The Pearson product moment correlation coefficient between the onset times of the sleep gates in the two conditions was 0.72 ($t=4.53$;, $df= 19$; $p<.01$). Since there were no differences between the distributions of onset times in the three experiments, the correlation was calculated from the pooled data.

5.3.4 Sleepiness structure adjusted to sleep gate

The fact that there was a within-subject stability of the timing of the nocturnal sleep gate made it possible to examine the 24-h structure of sleepiness adjusted to the individual sleep gate. To do so, each of the sleep histograms was phase-adjusted to the sleep gate, and then averaged across all subjects in each experiment. This was done separately for the two experimental conditions (Fig. 7-9). The adjusted histograms emphasized some trends in the data which were just noticeable in the unadjusted data. First, adjusting to the sleep gate greatly emphasized the abruptness of the nocturnal sleep gate, and the nadir in sleepiness immediately preceding the gate. From trials 13 to 7 before the gate, or from 4 to 2 hours before the gate, subjects were unable to fall asleep when instructed to do so and could easily resist sleep when instructed to avoid sleep. A second sleepiness nadir, occurring 24 hours after the first one, was evident in group 3, investigated for 36 hrs.

In experiments 2 and 3 which began at 0700 and 1100, the gate adjustment emphasized the secondary mid-afternoon sleepiness peaks occurring 7 hours $\pm$ 40 min before the major nocturnal sleep gate. The mid-afternoon peak in group 1, and the mid-afternoon peak during the second day in group 3, were less clear although in both experiments there was at that time a tendency for increased sleepiness, particularly in the RS condition.

The mean histograms adjusted to the sleep gate indicate

that the mid-afternoon peaks were more pronounced in the RS than in the AS condition. A repeated measures ANOVA (2 x 3 x 9) was performed to determine if this difference was reliable. The ANOVA was performed on the total amounts of sleep during the following time intervals: from trial 30 to trial 13 before the nocturnal sleep gate, in groups 2 and 3, and from trial 42 to trial 59 after the gate in group 1. These intervals correspond to the clock time period of 1300 to 1800 which contained the midafternoon peak. As expected the ANOVA revealed a significant interaction between sleep condition and hour of testing ($F=3.67$; $df=2,3$; $p<.02$).

5.4 DISCUSSION

Utilizing an ultrashort sleep-wake schedule has yielded detailed description of the 24-h structure of sleepiness. Although the overall general pattern is similar to the sleepiness pattern obtained by a much less frequent sampling procedure (Richardson et al. 1982), it contradicts the previous results by demonstrating that the onset of the nocturnal sleep period is abrupt, and not a gradual process as suggested by Richardson et al. In fact, this is the first time that it was shown that the timing of the onset of the nocturnal sleep period in subjects exposed to the geophysical zeitgebers is a stable individual characteristic, occurring as an 'all or none' phenomenon. Irrespectively of subjects' instructions to resist sleep or to attempt to fall asleep, or of the length of the sleep deprivation period, each subject

had his own sleep gate occurring within the wide range of 2100 to 0400 h. Over a two week period, the sleep gate for the same subject occurred at approximately the same time, suggesting that it is a dominant feature of the entrained sleep-wake cycle. This stands in contrast to Richardson's et al conclusion that the nocturnal sleep tendency is a continuously varying smooth function. However, since they sampled sleep tendency every 2 hours, they could not possibly detect the abrupt change in sleep tendency, as seen in the present study.

The present results also stand in contrast to the view that sleep time is less predictable than the time of waking up (Winfree 1982). This assumption was based on the fact that the decision when to go to sleep is more a matter of personal decision than wake-up time. Social obligations, studying for a next morning exam, or a fascination with a book may override the inclination to sleep. Sleep duration or wake-up time, on the other hand, was shown to be more closely related to the circadian phase of core body temperature (Czeisler et al. 1980), and therefore considered to be more predictable. The present results clearly show that for each individual there is a specific sleep gate from which time on a sleep-wake transition can be easily made.

The finding that the gating of nocturnal sleep is a stable individual characteristic also agrees with the experience gained in clinical sleep laboratories. It is not uncommon for some insomniac patients to find it very difficult to fall asleep if they pass their preferred

bedtime. Others, on the other hand, who suffer from the phase-delay syndrome (Czeisler et al. 1981) are unable to fall asleep before they reach their preferred bedtime in the early morning hours.

Just before the sleep gate, in both experimental conditions, subjects showed a pronounced decrease in sleep propensity. That is, at that time, subjects were unable to fall asleep when instructed to do so, and could easily avoid sleep when requested to remain awake. The pre-gate sleepiness nadir was clearly evident in experiments 2 and 3 in spite of the progressive sleep deprivation. This was particularly impressive in experiment 3 in which there was a second sleepiness nadir 24 hrs after the first one, in spite of the accumulation of 28-hr of total sleep deprivation plus 30-h of partial sleep deprivation under the ultrashort sleep-wake schedule. These observations lead to the conclusion that just before the sleep gate there is a 'forbidden zone' for sleep. That is, at that time there is a decreased likelihood for making a wake-sleep transition.

The conclusion that the time period just before the sleep gate is indeed a 'forbidden zone' for sleep, is supported by a different observation from our laboratory. In a patient suffering from a hypernychthemeral day (a sleep-wake cycle longer than 24 hrs) whose sleep-wake cycle has been followed for more than 4 consecutive years, there was a bimodel distribution of sleep onset times peaking at 2400 0200 and at around 1700. Just before the onset of the nocturnal sleep gate, there was a pronounced nadir in the

frequency of sleep onsets (at 2100-2300). This patient started a sleep period at 2300 only 6 times in 4 years, while he started a sleep period 95 times an hour later, at midnight (paper submitted to press). Thus, it can be concluded that just before the nocturnal sleep gate, preceding it by 2-4 hours, there is a 'forbidden zone' for sleep, during which sleep propensity reaches a nadir.

The structure of sleepiness observed in this study poses some difficulties to the theories attempting to model sleep-wake behavior according to homeostatic principles. The most recent theory (Daan et al. 1984) suggested a model which combined homeostatic principles and a single circadian oscillator. They assumed that a sleep promoting substance S is built-up during active waking and decreases during sleep. Sleep onset is triggered when S approaches an upper threshold and waking occurs when S reaches a lower threshold. These thresholds are suggested to be controlled by a single circadian oscillator. It is difficult to reconcile the structure of sleepiness during the second half of the day as revealed in the present study with a linear, or an even exponential accumulation of a sleep promoting factor. Progressive accumulation of such a substance would predict a parallel increase in sleepiness until the sleep threshold level is reached. Both the abruptness of the nocturnal sleep gate and the existence of a 'forbidden zone' for sleep just before the gate are incompatible with such an assumption. Although the Daan et al.'s model allows for shorter sleep-wake cycles under conditions which are sleep inducing and

therefore reduce the threshold for sleep, neither sleep deprivation, nor the specific experimental demands of AS and RS affected the basic structure of sleepiness. The stability of the sleepiness structure under all experimental conditions implicate a clock like rather than a homeostatic type of regulating mechanism. The close similarity between the temporal structure of sleepiness under the AS and RS conditions supports the interpretation that the variations in sleepiness are actively generated by a modulation of hypnogenic structures. Possibly an ultradian oscillator interacting with a circadian mechanism might be involved in this regulation, although the possibility of two coupled circadian oscillators cannot be excluded at this time. Actually, based on a two-oscillators model Kornauer and Strogatz demonstrated the existence of "forbidden zones" for sleep in data of free running subjects (Personnal communication).

It is interesting to note in this context that studies investigating the 24 hr structure of the propensity to wake-up from sleep by a progressive displacement of sleep, revealed a different pattern (Akerstedt and Gillberg 1981). The wake-up propensity was lowest between 1100 and 1900 and monotonically increased from 1900 until 0300. Subjective rating of sleepiness upon awaking from sleep tended to follow this pattern. The discrepancy between the 24-h patterns of sleep propensity and wake-up propensity supports the conclusion that sleep onset and wake onset are governed along different principles (Winfree 1982).

Previous studies linked variations in arousal to the 24 h temperature cycle (Czeisler et al. 1980; Zulley and Schulz 1980). Since temperature measurements were not taken in the present experiments, neither the 'forbidden zone' nor the 'sleep gate' could be correlated with variations in core temperature. But the accumulated literature on the 24h course of body temperature preclude a simple or direct sleepiness-temperature relationship. People who are neither extreme morning, nor extreme evening types, like all our subjects, as determined by a two-week sleep log, generally reach their peak body temperature during late afternoon. This time of deduced temperature peak coincided with the initial portion of the down trend of sleepiness in the present studies. The nadir in sleep propensity was delayed by 2 to 3 hours with respect to the deduced temperature peak, while the sleep gate was delayed by about 6 to 8 hours. Thus, two diametrically opposite gates occur along the descending slope of the temperature curve, separated from each other by only 2 to 4 hours. This makes the possibility that underlying changes in the phase of the temperature curve are responsible for the opposing gates unlikely.

Another interesting finding emerging from the present studies is the lack of significant differences between the amounts of total sleep in the AS and in the RS conditions. This indicates that when put in a sleep-inducing environment subjects cannot resist sleep, particularly when sleep deprived. This conclusion is not new. In 1960, Oswald showed that monotonic stimulation would induce sleep in n

This conclusion is not new. In 1960 Oswald showed that monotonic stimulation would induce sleep in normal volunteers even if the monotonic stimuli were painful electric shocks. Hartse et al. (1982) reported that differences between the results of the multiple sleep latency test and a comparable protocol of multiple sleep avoiding test (which is a comparable paradigm to the RS condition but with a sampling rate of once every 2 hrs) in normal subjects, disappear after one night of sleep loss.

There is no immediate explanation why the midafternoon sleepiness peak was more pronounced in the RS condition. Possibly dynamic interaction between the underlying variations in sleepiness and the specific experimental demands resulted in somewhat different time related trends of sleepiness.

5.5 Summary

Three experiments which utilized an ultrashort sleep-waking cycle were conducted to investigate the 24-h structure of sleepiness after one night of sleep deprivation under two experimental conditions: instructing subjects to attempt to fall asleep or instructing subjects to attempt to resist sleep. Six subjects participated in experiment 1. At 1900 they started a 15 min waking - 5 min sleep attempt, or 15 min waking - 5 min resisting sleep, until 1900 on the next day. Eight subjects were tested in a similar way in experiment 2, which started at 0700 after a night of sleep deprivation and

lasted for 24 hrs. Eight subjects were similarly tested in experiment 3 which started at 1100 after a night of sleep deprivation and lasted for 36 hrs until 2300 on the next day.

The results showed that in spite of the significance between groups differences in total sleep, the temporal structure of sleepiness was very similar in the three experiments. In each there was a bimodal distribution of sleepiness: a major nocturnal sleepiness crest and a secondary midafternoon sleepiness peak. These were separated by a 'forbidden zone' for sleep centered at around 2000-2200. The onset of the nocturnal sleep period (the sleep gate) was found to be a discrete event occurring as an 'all or none' phenomenon. Its timing was stable over a two week period, and independent of the specific experimental demands. there were no significant differences between the AS and RS conditions with respect to total sleep time or any of the sleep stages.

These results, which demonstrate structured variations in sleepiness across the nychthemeron are discussed in light of the recent modeling of sleep along homeostatic principles.

5.6 Figure Captions

Fig.1: Mean sleep histograms for the AS and RS conditions of experiment 1 (N=6). Legends of sleep stages are given in the top of the Figure.

Fig.2: Mean sleep histograms for the AS and RS conditions of experiment 2 (N=8). Legends of sleep stages as in top of Fig. 1

Fig.3: Mean sleep histograms for the AS and RS conditions of experiment 3 (N=8). Legends of sleep stages as in top of Fig. 1.

Fig.4: A Sleep histogram of a representative subject from experiment 1 in the RS condition demonstrating the occurrence of the sleep gate (G). Note the rhythmic occurrence of REM periods. Legends of sleep stages as in top of Fig. 1.

Fig.5: A Sleep histogram of a representative subject from experiment 2 in the RS condition demonstrating the occurrence of the sleep gate (G). Legends of sleep stages as in top of Fig. 1.

Fig.6: Scatter diagram of the timings of the sleep gate in the RS and AS conditions.

Fig.7: Gate adjusted sleep histograms for the RS and AS conditions of experiment 1. Legends of sleep stages as in Fig. 1

Fig.8: Gate adjusted sleep histograms for the RS and AS conditions of experiment 2. Legends of sleep stages as in Fig. 1

Fig.9: Gate adjusted sleep histograms for the RS and AS conditions of experiment 3. Legends of sleep stages as in Fig. 1

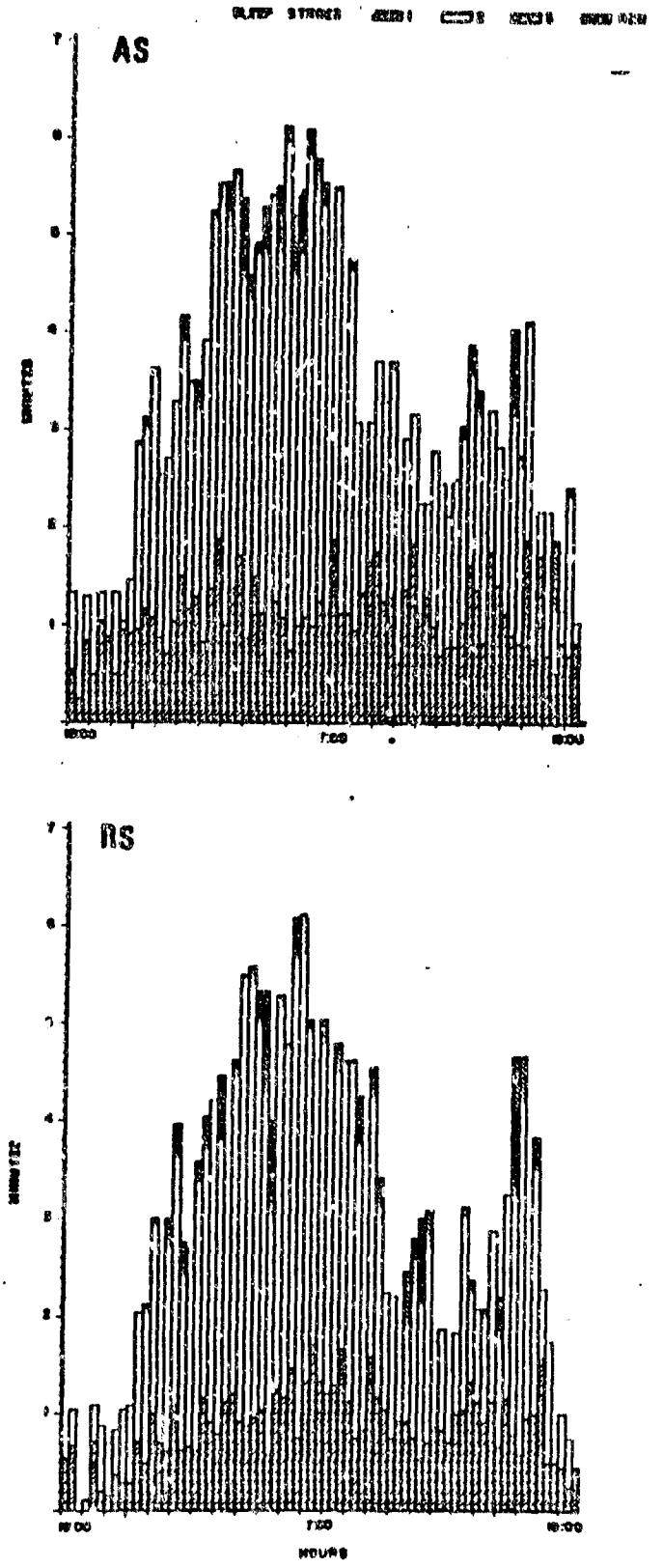

Fig. 1

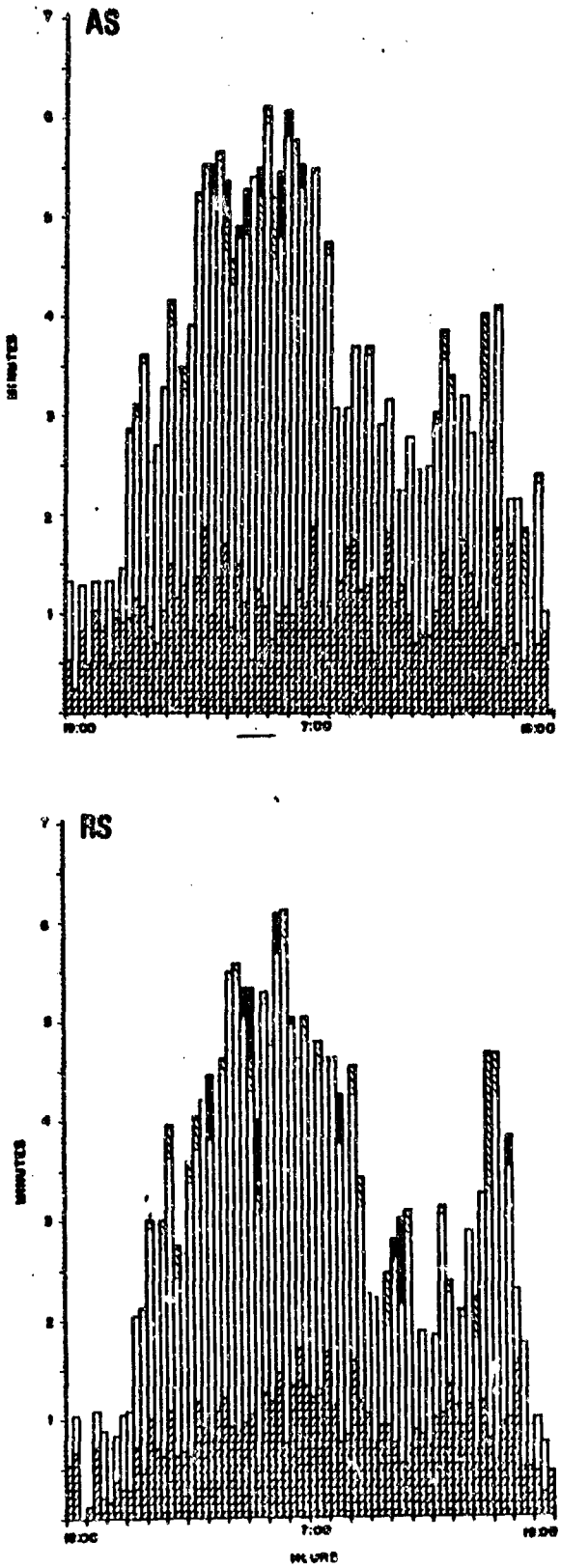

Fig. 2

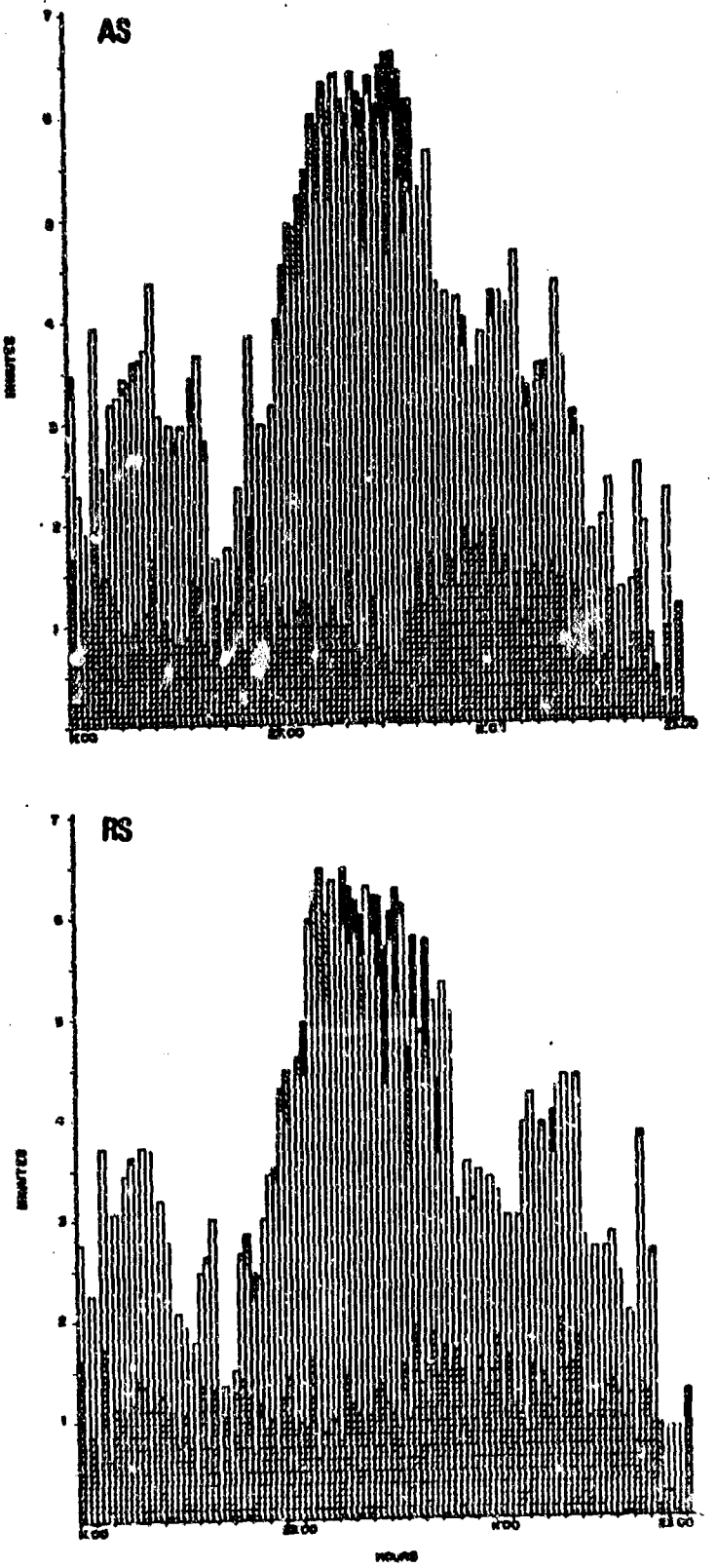

Fig. 3

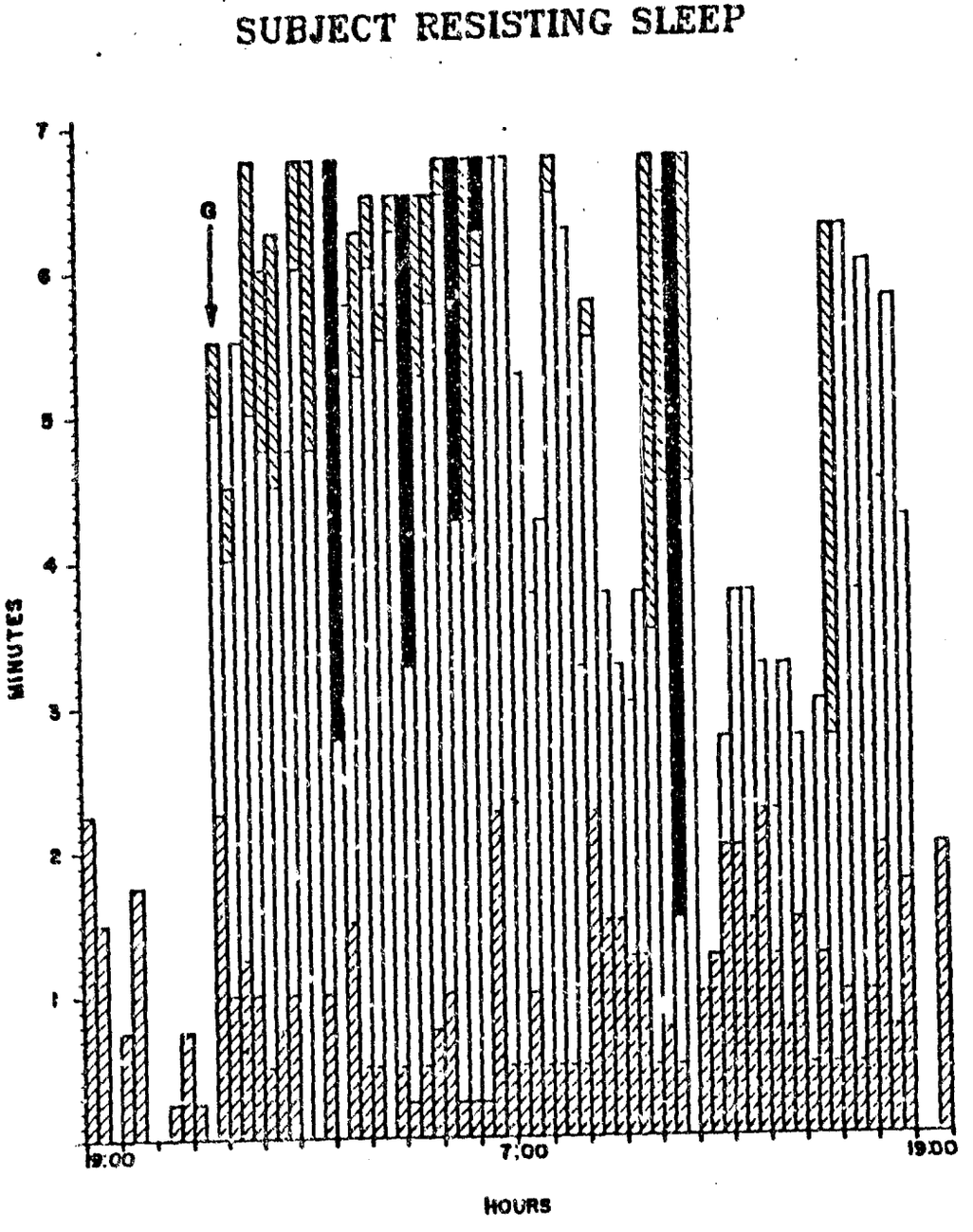
SUBJECT RESISTING SLEEP
MINUTES
7
6
5
4
3
2
1
G
19:00
7:00
19:00
HOURS

Fig. 4

SUBJECT RESISTING SLEEP

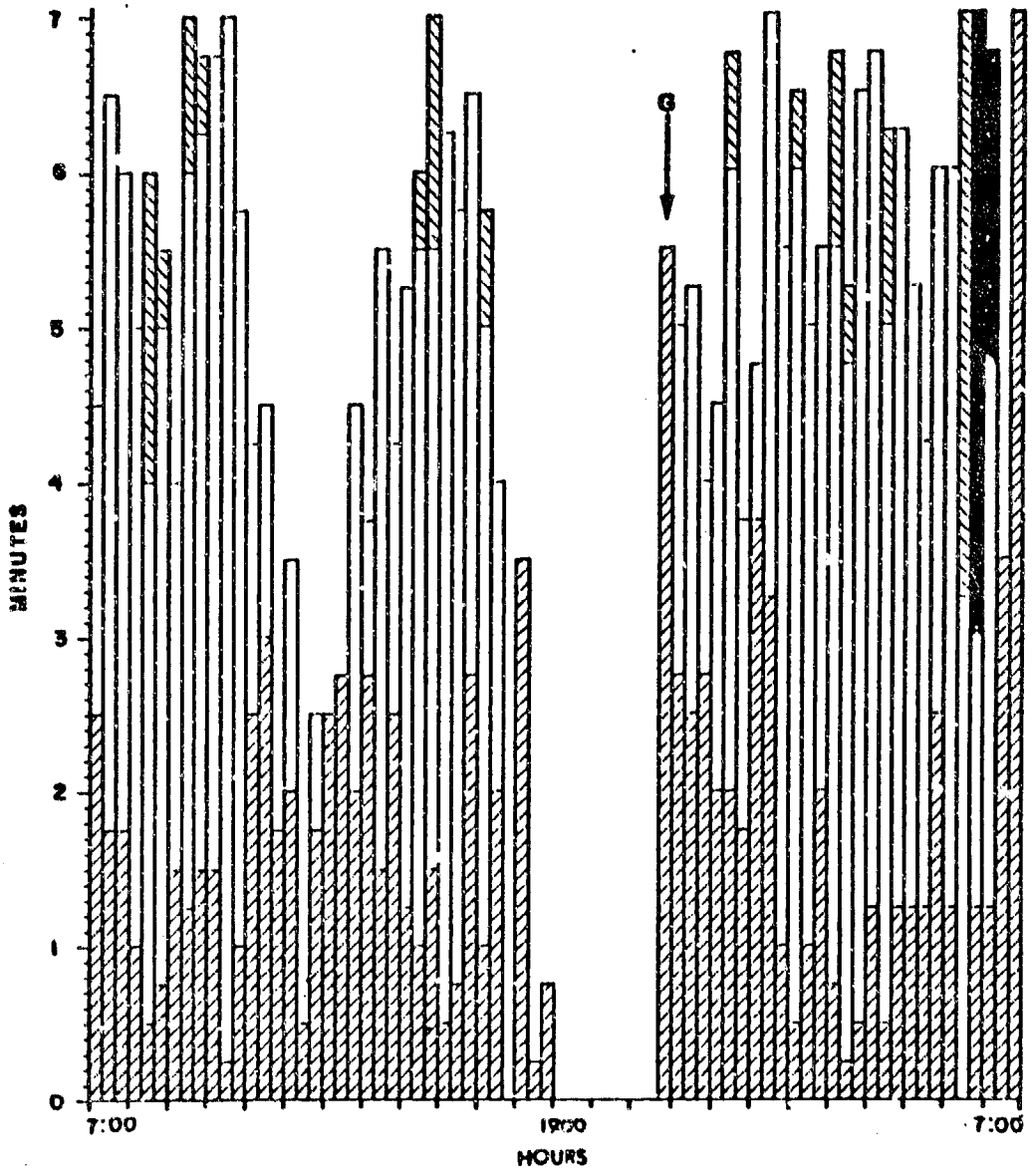

Fig. 5

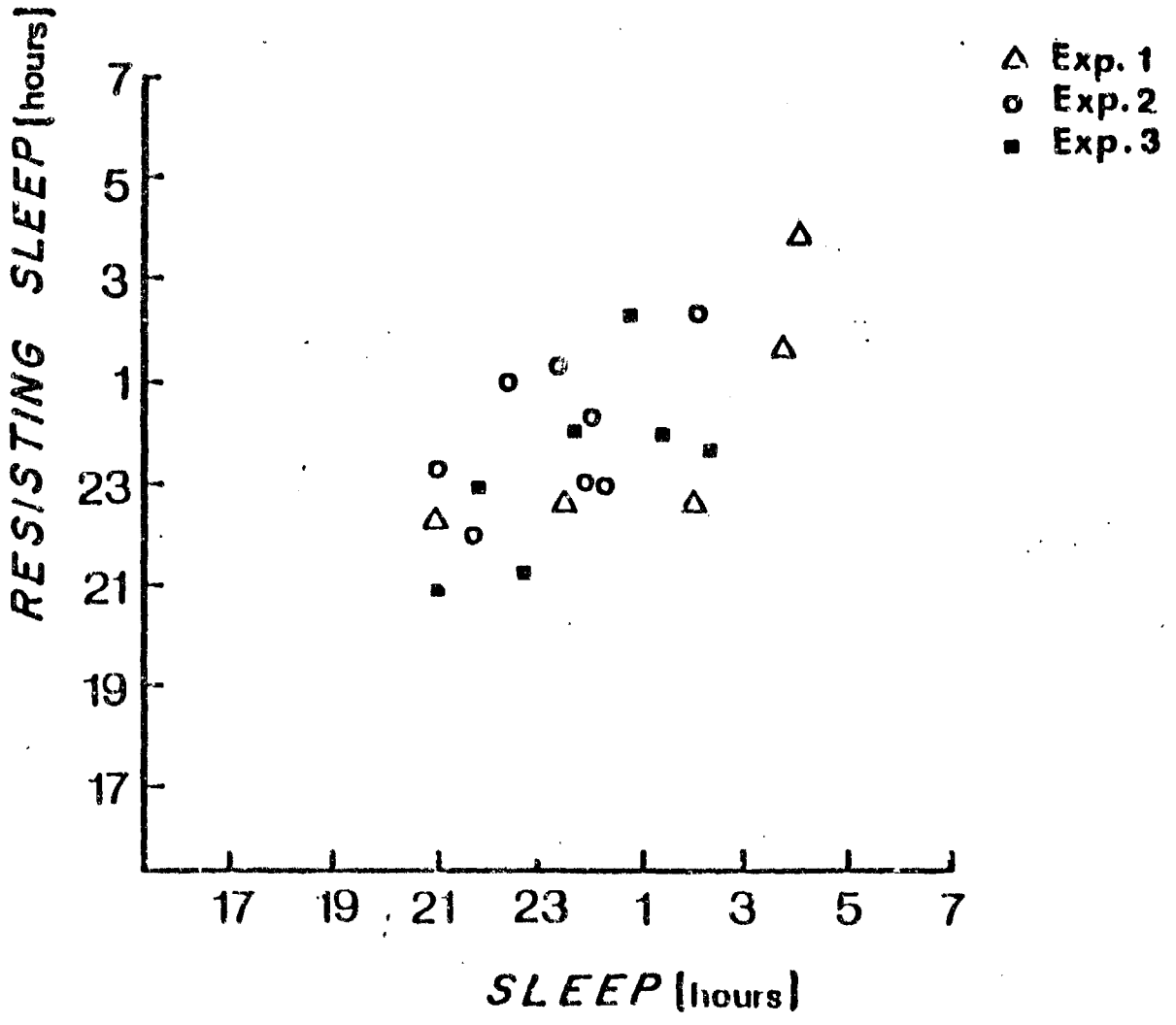
RESISTING SLEEP (hours)
7
5
3
1
23
21
19
17
17 19 21 23 1 3 5 7
SLEEP (hours)
Exp. 1
Exp. 2
Exp. 3

Fig. 6

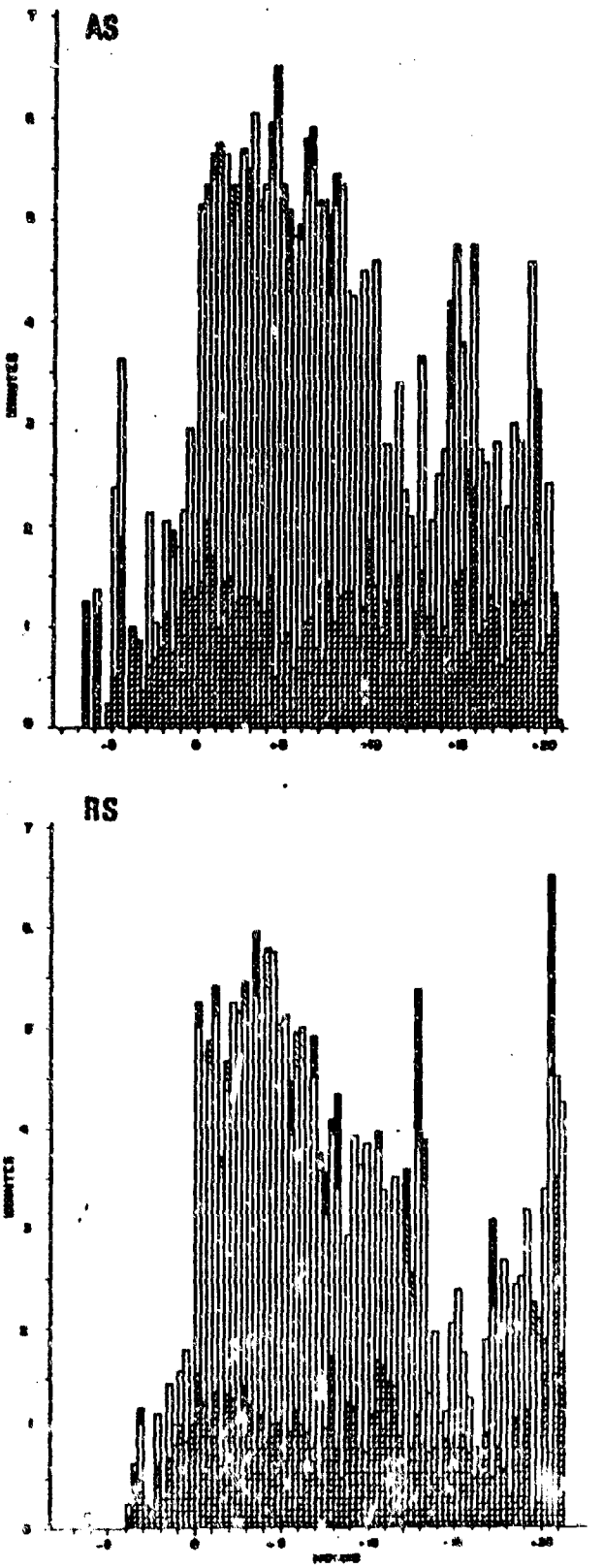

Fig. 4

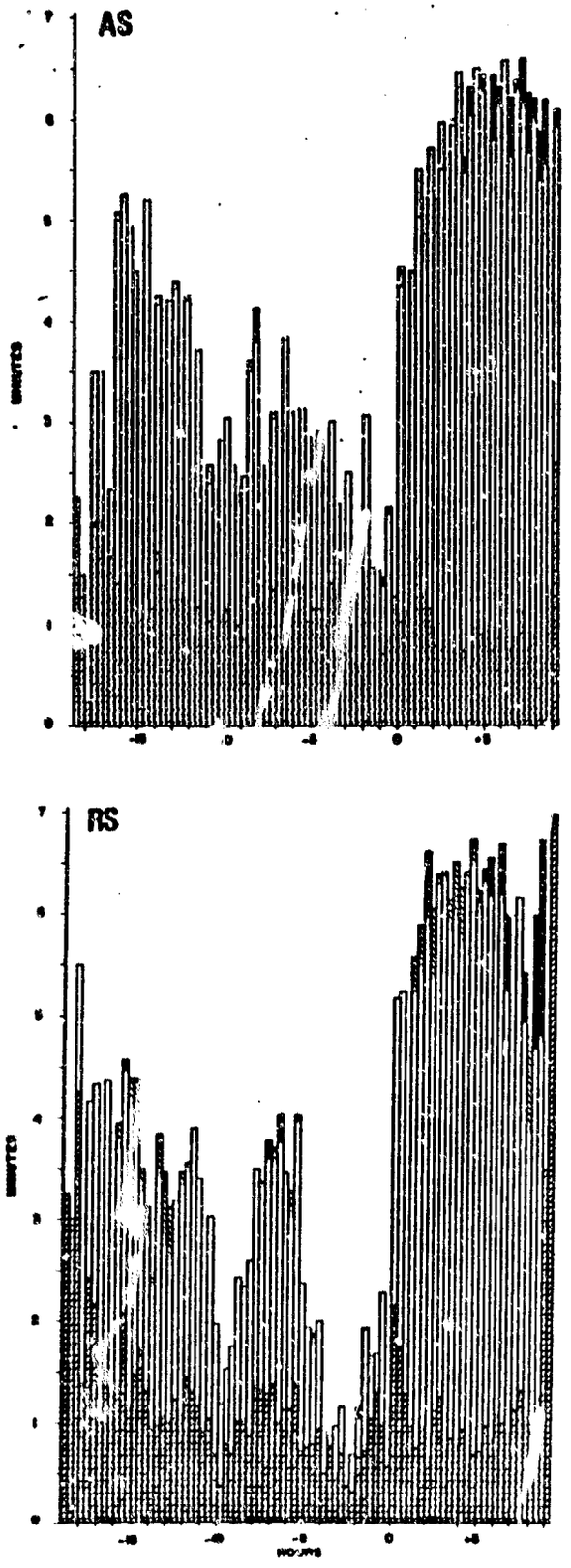

Fig. 8

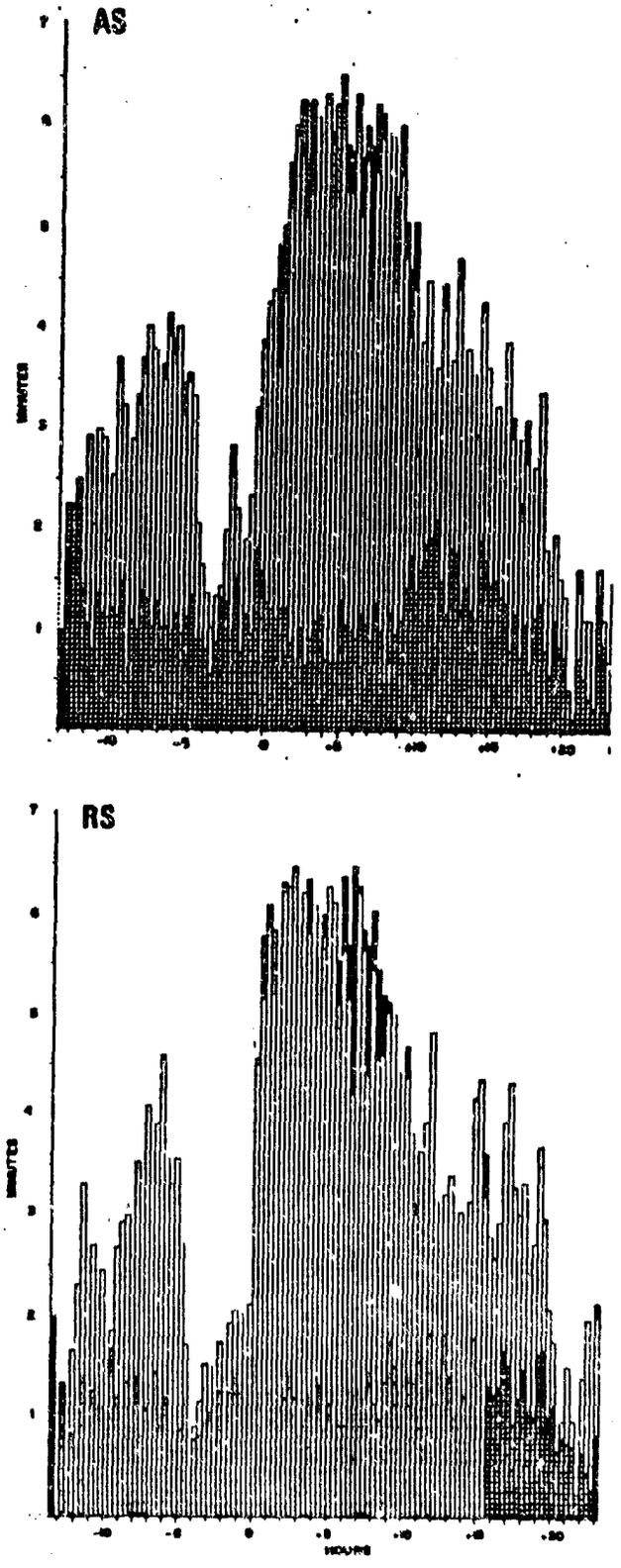

Fig. 9

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